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FIVE-YEAR STUDY OF CESAREAN SECTIONS IN MASSACHUSETTS*

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THE completion of the five-year study of cesarean sections in Massachusetts (1937–1941), sponsored by the Massachusetts Department of Public Health and the Section of Obstetrics and Gynecology of the Massachusetts Medical Society, allows one to summarize the findings and draw conclusions that might appear arbitrary if based on the statistics of any single year.

The statistics were compiled from replies to simple questionnaires sent to all licensed maternity hospitals, and yearly reports[1–5] have already been published.

The total number of births reported in Massachusetts during the five years was 333,731, of which 272,752 occurred in hospitals. The percentage of abdominal deliveries was 3.3. It is interesting to note the increase in the number of births occurring in hospitals each year.

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Over the five-year period, 11,030 cesarean sections and 87 hysterotomies were performed, a total of 11,117 abdominal deliveries. One abdominal pregnancy is not included in this report.

Table 1 shows that 45 per cent of the cesarean sections were emergency and 54 per cent elective operations, with the type not reported in less than 1 per cent. There were more low than classical type operations, but the ratio is not significant. The extraperitoneal and Porro operations account for about 1 per cent each. Sixty-five per cent of the patients were operated on before labor began, but 82 per cent were operated on with membranes unruptured.

The percentage of sections to the total number of deliveries has varied from 3.1 to 3.4 during the entire five years.

Table 2 gives the indications for the 87 hysterotomies. The large majority of these indications seem to be sound. A grave mistake occurred in 2 cases, in which the diagnosis was wrong and a

TABLE 1. *Summary of Cesarean Sections (1937–1941).*

DATA	1937	1938	1939	1940	1941	TOTAL	PERCENTAGE
Total births	63988	64112	64340	68261	73030	333731	
Births in hospitals	48966	50888	52697	56935	63266	272752	
Percentages of births in hospitals	76.5	79.4	81.9	83.4	86.6	81.7	
Cesarean sections	2082	2216	1984	2312	2436	11030	
Emergency	872	995	921	1051	1126	4965	45.0
Elective	1178	1196	1059	1247	1309	5989	54.3
Not reported	32	25	4	14	1	76	0.7
Type of operation:							
Low	1112	1206	1003	1235	1336	5892	53.4
Classical	870	934	898	992	1009	4703	42.6
Extraperitoneal	20	14	34	26	44	138	1.2
Porro	21	25	26	34	41	147	1.3
Not reported	59	37	23	25	6	150	1.4
In labor	681	744	616	757	806	3604	32.6
Not in labor	1333	1433	1302	1491	1601	7160	64.9
Not reported	68	39	66	64	29	266	2.3
Membranes ruptured	296	331	313	368	387	1695	15.3
Membranes unruptured	1697	1807	1600	1884	2014	9002	81.6
Not reported	89	78	71	60	35	333	3.0
Hysterotomies	24	16	12	21	14	87	
Percentages of abdominal deliveries	3.2	3.4	3.1	3.4	3.3	3.3	

hysterotomy was done unnecessarily. It is doubtful if the indications "severe bleeding" and "placenta previa at three and a half months" were justifiable.

In Table 3 are recorded the types of anesthesia. Nitrous oxide, oxygen and ether is the type most commonly used. The only other point to be noted is the marked increase in the use of spinal anesthesia. The widespread use of local anesthesia in cesarean sections has never been received with favor in this community. It is unfortunate that 11 deaths have to be recorded as due to the anesthetic.

In each year's study, operations resulting in the deaths of babies have been analyzed under emergency and elective sections. In the emergency classification, 741 babies were lost, and in the elective group 245—a total of 986. To analyze all the indications for the five years would be a lengthy process, but it must be noted that almost 50 per cent of infants were lost because of placenta previa or separated placenta. One questions the advisability of many of these operations, for they were done at six or seven months' gestation and could undoubtedly have been managed satisfactorily by vagina.

It must also be noted that many babies died or were stillborn following elective sections. It is fair to assume that some would have died following a normal delivery, but when a study of the questionnaires reveals that in many of the elective sections the babies were very small and obviously premature, it is evident that the time of operation was badly chosen.

I shall speak later of the questionable judgment shown in performing sections on known monstrosities, which could well have been delivered by vagina.

Table 4 records the indications for sections for the five-year period. Previous section, of course, tops the list. In this group, many babies were lost from prematurity because the operation was done by election too early in the gestation or because labor had begun prematurely. These possibilities must be kept in mind in all repeat sections. The next largest group comprises patients with a contracted pelvis or disproportion. In many cases, this indication was probably not justified, for it was frequently noted that the baby weighed under six pounds, and often the patient did not even go into labor.

About the next two indications, placenta previa and separated placenta, one can only say that there was bleeding. Many of these babies were lost. The number of patients operated on at six and a half to seven months raises the question whether all these sections were necessary. The frequency of fetal malformations in cases of

TABLE 2. *Indications for Hysterotomy.*

INDICATION	1937	1938	1939	1940	1941	TOTAL
Cardiac disease	8	7	5	5	6	31
Hypertension or nephritis	4	2	3	9	7	25
Toxemia	2	0	1	0	0	3
Nephrolithiasis	1	0	0	0	0	1
Hydatid mole	2	1	0	0	0	3
Mental disease	1	1	2	2	0	6
Tuberculosis	1	1	0	2	0	4
Neurologic disease	0	2	1	1	0	4
Placenta previa (3½ months)	1	0	0	0	0	1
Severe bleeding	0	0	0	0	1	1
Acute polyhydramnios	1	0	0	0	0	1
Erythroblastosis in previous pregnancy	0	1	0	0	0	1
Fibroid uterus	0	1	0	0	0	1
Separated symphysis	0	0	0	2	0	2
Error in diagnosis	2	0	0	0	0	2
Not reported	1	0	0	0	0	1
Totals	24	16	12	21	14	87

TABLE 3. *Types of Anesthesia.*

ANESTHETIC	1937	1938	1939	1940	1941	TOTAL
Nitrous oxide, oxygen and ether	1343	1533	1255	1355	1244	6730
Ether	348	260	194	261	272	1335
Cyclopropane	103	159	136	144	166	708
Spinal	85	106	185	396	641	1413
Nitrous oxide and oxygen	81	19	18	5	8	131
Avertin and nitrous oxide, oxygen and ether	50	29	29	27	15	150
Local	37	34	22	32	62	187
Other	21	50	135	70	15	291
Not reported	14	26	10	22	13	85
Totals	2082	2216	1984	2312	2436	11030

placenta previa makes an x-ray examination advisable before operation for this indication.

The majority of operations in the next group were because of an increasing, severe toxemia. Many of these pregnancies were allowed to continue too long, with the result that some of the necessity for operation. If the cervix has been amputated, it should be known before labor begins, and an elective section performed.

Under "associated medical condition," the largest group was for cardiac conditions. The necessity or even the advisability of operating in many

TABLE 4. *Indications for Cesarean Section.*

INDICATION	1937	1938	1939	1940	1941	TOTAL
Previous section	576	635	608	726	722	3267
Contracted pelvis or disproportion	539	477	336	350	491	2193
Placenta previa	183	175	215	211	237	1021
Separated placenta	77	104	86	109	106	482
Toxemia	123	125	78	107	114	547
Eclampsia	10	9	11	15	8	53
Malposition of baby	68	110	79	113	109	479
Dystocia	94	202	220	279	246	1041
Previous surgical operation	45	43	36	56	50	230
Associated medical condition	59	61	51	51	46	268
Previous obstetric disaster	64	47	52	73	76	312
Elderly primipara	48	70	58	57	58	291
Obstructing tumor	15	18	24	21	44	122
Fetal distress	5	7	6	7	11	36
Prolapsed cord	9	3	4	4	7	27
Following operative attempts	3	7	7	8	10	35
By request, or for sterilization	27	17	10	9	10	73
Ruptured uterus:						
Spontaneous	1	1	2	2	2	8
Previous section	1	7	6	2	4	20
Abnormality of genitourinary tract	3	4	2	3	7	19
Twins	2	0	0	3	5	10
Postmaturity	8	4	3	8	8	31
Malformation of fetus	4	1	4	9	1	19
Varicosities of vulva	1	2	0	1	1	5
Unengaged or floating head	5	0	0	3	0	8
Congenital dislocation of femur	0	1	0	0	1	2
Fractured pelvis	0	6	0	0	1	7
Error in diagnosis	2	0	1	0	1	4
Surgical emergency	0	0	1	0	1	2
Early rupture of membranes	0	0	0	2	0	2
Young primipara	0	0	0	1	0	1
Multiparity	2	0	0	0	0	2
Prolapse of uterus	2	0	0	0	1	3
Premature labor	3	0	0	0	0	3
Feeblemindedness	1	0	0	0	0	1
Social and economic reasons	0	3	0	0	0	3
Abdominal hernia	0	1	0	0	0	1
Obesity	0	1	0	0	0	1
Desire for living baby	0	1	0	0	0	1
Frequent miscarriages	0	1	0	0	0	1
Bizarre	55	25	32	19	19	150
Not reported	47	48	52	63	39	249
Totals	2082	2216	1984	2312	2436	11030

mothers died. One cannot help believing that if there were more careful prenatal care, fewer patients would be operated on for this indication.

It is surprising that eclampsia should have been the indication in 53 cases. Eleven mothers (21 per cent) and 9 babies were lost, a very high mortality.

An increasing number of sections occurred for breech presentation. Many of the patients were multiparas, and the babies were of moderate size. It is obvious that when any malposition appeared, there was little attempt to manage it by vagina.

Under "dystocia," I have included the so-called "test of labor" cases. In the latter part of the study, there were many more attempts to give the patient a test of labor. Some cases of so-called "dystocia" had no labor whatsoever.

The majority of sections because of previous surgical operations were for repairs of the perineum or cervix. Again, there is no way of judging the cardiac cases is open to question, for it is known that such patients often have short, easy labors. With careful management, they may be better delivered by vagina. Careful consideration of all factors is necessary before the decision to operate on a cardiac case is made.

Kidney complications, central-nervous-system disease and tuberculosis were some of the other medical indications. Because one clinic believes that many diabetic patients should be delivered by section, the frequency of this indication has increased.

If operation seems indicated with a history of previous obstetric disaster or difficult operative delivery, it should be elective or should follow a short test of labor under careful supervision. Labor for eighteen to twenty-four hours without progress, followed by an emergency operation, should never occur with this indication.

It is impossible to judge whether all the elderly primiparas required sections. If the indication is real, however, it should be an elective, not an emergency, section.

Obstructing tumors should be discovered by careful prenatal examination, and operation for this indication should also be elective, not emergency, as often happened.

Only 3 babies were lost following section for fetal distress, and 5 for prolapsed cord. One wonders whether the fetal distress was real. Undoubtedly, cesarean section for prolapsed cord will save a certain number of babies, but the infant mortality rate for this indication was 18 per cent.

A section following operative attempts at delivery is a serious procedure. Although a certain number of mothers and babies may be saved, the mortality rate is high. In this group of 35 patients, 10 mothers and 11 babies died.

I cannot consider the next indication justifiable. Yet 73 operations were done at the request of the patient or for sterilization.

Comment on the indication "ruptured uterus" is unnecessary, for the patient with this condition must, of course, be operated on as quickly as possible.

Under "abnormality of genitourinary tract," a surprising number were bicornate uteri.

"Twins" was the recorded indication in but 10 cases. Many other cases of twins occurred, but the operation was for some other indication. In a few of these, "disproportion" was given as the indication, no x-ray film was taken, and twins were delivered. There were 2 cases of triplets.

The indication, "postmaturity," is specious, especially when, as happened several times in this series, a baby weighing 7 pounds or less was delivered.

For the next indication, "malformation of fetus," 19 sections were done with a known malformation present. Accompanying this indication, there was no statement that the patient had a contracted pelvis. Unless there is some other indication, a section on a known malformation seems to me entirely unwarranted. The surgeon should ask himself whether he would like his wife to be operated on for a known malformation of the fetus incompatible with life; I think the answer is clear. No religious dogma demands that a section be done under these circumstances. In 1 case in which a section was done, the hydrocephalic head was so large that it had to be punctured before it could be removed from the uterine incision.

Under "unengaged or floating head," 8 cases were operated on without any labor, again an unjustifiable procedure.

If congenital dislocation of the femur has so narrowed the pelvis that a baby cannot be delivered through it, that indication is of course real. The same thing is true of fractured pelvis.

It is unnecessary to go through each of the other indications. It is seen at once that some are questionable. Under the heading, "bizarre," 150 cases have been grouped. A few will serve as examples: "pregnancy at term," "Braxton-Hicks contractions," "highly nervous state of the mother," "pain in right hip" and "threatened placenta previa." I might go on with many other equally bizarre indications.

Table 5 presents a summary of the maternal deaths. In making this study concurrently with the five-year study of maternal mortality in Massachusetts, it has been possible to obtain data concerning the deaths from cesarean section from the

TABLE 5. *Summary of Maternal Deaths in Cesarean Sections.*

DATA	1937	1938	1939	1940	1941	TOTAL	PERCENTAGE
Total maternal deaths	66	60	54	41	52	273	2.46
Emergency	51	39	45	33	36	204	74.7
Elective	15	21	9	8	16	69	25.2
Type of operation:							
Low	29	26	24	15	16	110	40.0
Classical	29	24	26	20	30	129	47.4
Extraperitoneal	6	2	2	2	3	11	4.0
Porro	2	3	2	4	3	14	5.1
Not reported	4	5	0	0	0	9	3.0
In labor	31	25	29	22	20	127	46.5
Not in labor	32	30	25	19	32	138	50.5
Not reported	3	5	0	0	0	8	2.9
Membranes ruptured	13	18	15	17	13	76	27.7
Membranes unruptured	47	37	39	24	39	186	68.0
Not reported	6	5	0	0	0	11	4.0
Babies:							
Living	43	43	42	36	40	204	73.8
Dead	23	17	12	8	12	72	26.1

maternal-mortality investigations of the same cases, which were made by physicians in personal interviews. This has given a much more accurate picture of the cesarean deaths than the questionnaires alone would have revealed. As a result, in many cases, the causes of death given on the questionnaires have been found inaccurate, and the causes of death from the maternal-mortality schedules have been accepted.

Over the five-year period, there were 273 deaths. Of these, 74.7 per cent followed emergency operations, and 25.2 per cent followed elective sections. Fewer patients were lost following low than classical sections, but I am inclined to think that the reason the classical type was performed more frequently is that it can be done more quickly in an emergency.

Eleven patients, or 4 per cent, died following extraperitoneal operations. Some were undoubtedly neglected cases. Others were under observation by competent physicians the entire time, and yet they were allowed to remain in labor so long that the physicians were forced to do extraperitoneal operations resulting in death. In this operation, there is danger of opening the bladder or cutting a ureter, as was done in this group.

Five per cent of the deaths followed a Porro operation. The majority of these were done for fibroids or an atonic uterus.

Table 6 shows the causes of the maternal deaths. The total of 273 deaths gives a mortality rate of 2.46 per cent, a remarkably good record. I doubt if any other state has excelled it. Analysis of the deaths, however, shows some disturbing facts.

TABLE 6. *Causes of Death in Cesarean Sections.*

CAUSE OF DEATH	NO. OF CASES	PERCENTAGE OF TOTAL
Sepsis	106	38.8
Hemorrhage	32	11.7
Embolus	28	10.0
Shock	25	9.1
Cardiac	17	6.2
Anesthesia	11	4.0
Eclampsia	10	3.6
Acute respiratory infection	8	2.9
Toxemia	7	2.5
Anaphylaxis	6	2.1
Intestinal obstruction	4	1.4
Acute pulmonary edema	4	1.4
Premature separation of placenta	3	1.0
Miscellaneous (1 each, chronic nephritis, toxic purpura hemorrhagica, thyrotoxicosis, superior mesenteric thrombosis, scarlet fever, acute yellow atrophy, anuria, uremia, surgical emergency, carcinoma, no adequate cause and not reported)	12	4.3
Total	273	

One hundred and six patients, or 39 per cent, died from sepsis. Of these, 24, or 23 per cent, were elective sections. It was in these cases that most of the changes were made in the causes of death. The following history is typical. Immediately following operation the patient's temperature and pulse rose. There was distention, vomiting and delirium, and death followed. The questionnaire gave the cause of death as "toxic myocarditis, vasomotor collapse and paralytic ileus." Interview by the investigator in the maternal mortality study brought out the true facts. The desire to cover up the diagnosis of sepsis led to improper certification of the cause of death.

Hemorrhage accounted for 32 deaths, or 11 per cent. The majority of these followed section for either placenta previa or separated placenta. Often, when a section has been done for separated placenta and bleeding continues, a quick decision must be made whether or not to remove the uterus to save the patient. In several cases, the uterus failed to react, and yet the surgeon did not remove it. Several patients died from postpartum hemorrhage two or three hours after operation, which suggests a lack of careful nursing supervision.

There were 28 deaths, or 10 per cent, from embolus. Some of these patients had a definitely septic chart, and although the death was undoubtedly due to embolus, it might well be classed in the septic group.

Shock was given as the cause of death in 25 cases, or 9 per cent. It was frequently difficult to determine whether death should be assigned to shock or to hemorrhage.

In many of these fatal cases, no donors were provided for transfusion. In one case, the operation was done by a general surgeon; the patient bled profusely during the operation and continued to bleed afterward; yet the surgeon left the hospital as soon as possible after the operation, and made no attempt to stop the bleeding. The local physician was unable to cope with the situation, and the patient died from post-partum hemorrhage. On the whole, however, the use of transfusion has been gratifying and has undoubtedly saved many lives.

The death of 11 patients, or 4 per cent, from anesthesia is a serious criticism. Careful anesthesia must be provided in all cases.

I have already spoken of the cases of eclampsia and toxemia. Some of the latter were elective sections, and the patients should not have been lost.

The deaths from acute respiratory infections were accidents and could have been avoided only by the most meticulous care.

Of the anaphylactic deaths, 1 resulted from what is called "pituitary shock." The other 5 were classified as due to improper matching of blood. That statement must now be revised in the light of the recent work on the Rh antibody.

It is fair to state that these reactions may have been due to this newly discovered factor.

That 4 deaths should have been caused by intestinal obstruction was undoubtedly due to lack of keen observation on the surgeon's part.

In the cases of acute pulmonary edema, it is noted that the patients were operated on after they had been in labor a very long time.

Although only 3 deaths were assigned to premature separation of the placenta, this complication occurred in some cases in which the death was assigned to other causes.

The miscellaneous list of 12 causes of death needs little explanation. The scarlet-fever case was apparently true scarlet fever, and the surgical emergency was one in which there was an aneurysm of the splenic artery, which ruptured. In the death assigned to acute yellow atrophy, the patient had an unusual blood condition and bled from the gums, stomach and other mucous membranes. The uremic case was one in which the patient had had one kidney removed, developed toxemia during pregnancy and gradually failed, with an increasing nonprotein nitrogen. The death assigned to carcinoma was that of a patient whose entire pelvis was involved with cancer, the section being done to save the baby.

* * *

This is the largest series of cesarean sections ever reported from one community, so far as I have determined, and the results are remarkably good. Examining them superficially, one could not criticize them. But on analysis, one must conclude that many unnecessary cesareans were done. Because of this, the number of repeat sections will continue to increase and will always give a certain number of deaths. The same criticism is true regarding the indication "contracted pelvis or disproportion." There is no doubt that many physicians do not carefully study each case. Cesarean section is an easy way out of a difficulty, but it often has serious consequences for the mother in future pregnancies.

The fact that nearly 40 per cent of the patients died from sepsis is a very serious criticism of the medical profession. It shows conclusively that there was either error in technic or in the choice of a method of delivery. In some cases, when a low or classical section followed a long labor, sulfanilamide was used in the abdomen. Many of these patients eventually recovered, even though the convalescence was stormy. It is conceivable that this will become a routine treatment in emergency cesareans with a possibility of infection, and that the percentage of septic deaths may thereby be greatly reduced.

The maternal mortality rate for these cesarean sections, 2.46 per cent, was low, but when one realizes that in the maternal-mortality study, cesarean section figured in nearly 20 per cent of the deaths, it is evident that such deaths are a potent factor in keeping the State maternal mortality higher than it should be.

In closing, I want to thank the hospitals that co-operated so splendidly in making out their questionnaires. It added a great deal to the work of their record rooms. In nearly all cases, it was well done, and the true facts were reported. Only in a few cases was there lack of co-operation; this occurred chiefly when the physician obviously wished to cover up poor obstetrics.

Under the new law making the Department of Public Health responsible for licensing all hospitals, and with an investigator checking their records and procedures, one can look forward confidently to even better results.

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References

1. DeNormandie, R. L. Cesarean section in Massachusetts in 1937. *New Eng. J. Med.* 219:871-878, 1938.
2. *Idem.* Cesarean section in Massachusetts in 1938. *New Eng. J. Med.* 223: 45-50, 1940.
3. *Idem.* Cesarean section in Massachusetts in 1939. *New Eng. J. Med.* 224: 963-971, 1941.
4. *Idem.* Cesarean section in Massachusetts in 1940. *New Eng. J. Med.* 227: 121-128, 1942.
5. *Idem.* Cesarean section in Massachusetts in 1941. *New Eng. J. Med.* 227: 499-502, 1942.

TRAUMA OF THE SKIN DUE TO WARTIME ACTIVITIES*

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BOSTON

AN emergency came, and we were found wanting. It must never happen again! Preparedness must be our watchword, not only for defense but also for offense when the opportunity arises. Physicians must be prepared to relieve and prevent injuries due to wartime activities. No longer may the specialist be content to confine his study to the immediate realm of his specialty — he should be trained for any emergency. While lay persons, because of their attendance at various courses, are glibly talking of poisonous gases and first aid, many medical men still remain ignorant of some of the essentials of both.

Care of the skin is vital at present. During the Spanish Civil War, cutaneous disease caused untold suffering and disability in civil and military populations, and helped to decide the outcome of the war. The same diseases played a great part in World War I. Medicine has learned tragic lessons from these experiences, but there are still many pitfalls to be avoided. It has been stated that this war will be one of industry and production. The producers must therefore be kept healthy — that is, occupational diseases, of which dermatoses comprise 60 per cent, must be prevented. President Roosevelt, in a recent newspaper interview, expressed his alarm over this aspect of industry: 460,000,000 man-days of work lost; 101,800 persons killed; 350,000 permanently disabled — 1 out of every 14 workers, most of them from twenty-one to forty-four years of age, or enough to build several battleships or equip several regiments. The President hoped that everyone would reflect on these figures, so that the situation might be remedied or at least improved, and he must have had medical men particularly in mind.

New plants are being equipped, and old plants are experimenting with substitutes, new chemicals and chemical processes. Speed is essential to fill contracts within specified time limits, but reckless haste results in disaster. Overcrowding in factories and in the housing of workers must be avoided. Although the older and larger plants are properly prepared, others lack adequate preventive methods, such as means of ventilation and exhausts, and many factories have no facilities for the personal hygiene of the workers and no medical supervision. Too few physicians are properly trained to cope with these problems, and still fewer have sufficient knowledge of industrial poisons. Recent events created a national emergency that subordinated the welfare of the individual to that of the Nation. Nevertheless, it behooves the physician to watch the individual, for each person affected may be a warning of the imminence of an epidemic of disabling dermatitis of physical, chemical or infectious origin, whether in civil, industrial or military life. At present, it may seem strange to assert that, of the three groups, the industrial is the most important. Yet it is reasonable to believe that the loss of one key worker may delay the necessary equipment of many fighters. Hilliard[1] points to the need of a public-health program and states that there are the same possibilities of reducing sickness among industrials who congregate daily as there are in preventing diseases in the general community. At present, the most essential wartime industrial activities are the manufacture of garments, machinery, tools and explosives, aircraft production and shipbuilding.

OCCUPATIONAL DERMATOSES

Recognition of an industrial dermatitis may often prevent similar occurrences in military life. For example, an outbreak of dermatitides in a plant making raincoats was shown to be due to free sulfur. To hasten production, the rubber was semicured, resulting in the release of the free sulfur and causing erythema and intense itching of the skin of many employees. Such coats would have irritated the soldiers wearing them.

In another plant manufacturing rubber goods, it was found that the old rubber cement could not stand the intense heat essential for the rapid turnover, and a new cement was therefore introduced. The cementers encircled a wad of the rubber cement around their right index fingers and applied it to the seams and edges. They suffered a dermatitis consisting of edema, desquamation and fissuring of the fingers, with extension of a sensitization eruption over the hands and arms, with resulting disability and loss of production.

In April, 1940, I investigated the cause of an epidemic of cutaneous diseases among boys and men wearing new fabrics. Each eruption started with marked erythema and edema of the penis and then of the scrotum; later, vesicles and wheals appeared on the loins, and the rash frequently be-

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came generalized. It was accompanied by such intense pruritus that one patient became temporarily violently insane, so that it was necessary to commit him to a psychopathic hospital. The

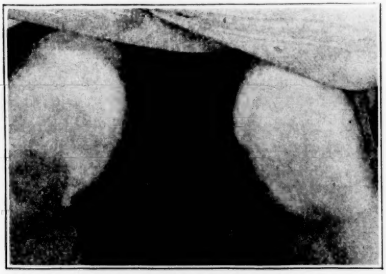

FIGURE 1. *Painful Heels due to Cement in Shoes.*

The skin appears white and sodden, like boiled meat. A physician suffering from this disturbance was willing to undergo a sympathectomy for relief; change of footwear avoided this.

causative factor was found to be a new synthetic resin finish that had previously affected the employees in textile industries. The same finish had

FIGURE 2. *Leather Dermatitis.*

An eruption on the distal half of the dorsum of the foot should suggest such a diagnosis, rather than that of a fungus infection.

been applied to cloth intended for the army personnel, but was withdrawn and not issued after this investigation. It would have caused more disability than a horde of Japanese.

In another textile plant, two workers handling indigo cloth for sailors' blouses suffered a dermatitis from a change of the formula. Sensitivities to these clothes should be kept in mind, for they are already appearing in the coast guard and naval personnel.

Shoe workers are still suffering from hazards incident to their trade, and eruptions are occasionally seen from footwear (Figs. 1 and 2). These patients are usually treated for fungus infections, owing to mistakes in diagnosis.

Among machinists and their helpers, dermatitides from cutting oils have become unnecessarily numerous (Fig. 3). The cutaneous manifestations have been of two types: mechanical interference with bodily function, that is, folliculitis; and the reaction of hypersensitivity, that is, eczema. In the first group, the skin becomes dry and scaly,

FIGURE 3. *Folliculitis from Cutting Oils.*

The skin shows a breaking off of the hairs, papules and pustules.

and the lanugo hairs break off at the point of emergence from the follicles, leaving a black-dot appearance; comedones result from the collection of epithelial debris and dirt; later, acuminate papules, which become secondarily infected and form pustules, furuncles and large abscesses, appear. These heal with scars varying from smooth pigmented atrophy to large keloids. In the second group, the eczematous dermatitis is localized or generalized. Men assembling small parts work with their hands close together and suffer erythematous, dry, fissured eruptions of the hands, because of constant contact with the drying oil. In the assembling of large parts, the oil splashes on their arms and clothing, and the eruption appears on the arms, especially in the antecubital spaces and on the abdomen and the anterior aspects of the thighs. Admixtures such as terpenes, creosols, acids and alkalies are usually the sensitizers. Minute pricks from metal chips produce portals of entry for bacteria, with resulting pyodermia. Cancers are infrequent.

Similar eruptions appear in workers exposed to chlorinated naphthalene, such as men handling

cables containing halowax used for insulation of wires. This is especially true in poorly ventilated places, as in ships under construction. In these exposures, the workers must be watched because of the danger of systemic poisoning. Six electricians working in a submarine developed yellowish, wax-like milia on the sides of the neck, the lobes of the ears and the cheeks, accompanied by comedones, papules, pustules and nodules (Fig. 4).

FIGURE 4. *Acneform Eruption from Halowax. The skin shows yellowish, wax-like milia, comedones, papules, pustules, and nodules.*

Two had similar lesions on the forearms and thighs. No systemic disturbances were discovered after careful physical examination and laboratory studies.

In the manufacture of explosives, trouble rarely occurs among the older employees, but new workers, who may not be immune and who do not realize the danger in handling the various materials, sometimes neglect to take the proper precautions. Dinitrotoluol and the nitrates, especially ammonium nitrate, are cutaneous irritants, and the workers who mix them develop a dermatitis. Fulminate of mercury is used in the making of detonators and percussion caps. Workers exposed to this compound may show signs of mercurial absorption, and those who perspire freely may suffer an affection of the skin of the neck, behind the ears, of the face and in the antecubital spaces. There is inflammation of the nasal mucous membrane, the skin is discolored, and there may be shallow ulcerations at the tips of the fingers that are relieved by 10 per cent sodium hyposulfite. Tetryl and trinitromethyl nitramine also cause eruptions among new workers. An eczematous dermatitis appears at the edges of the respirators and the volar surfaces of the forearms. Two types of cutaneous eruptions may appear: mechanical interference because of the penetration of the dust into the follicles and the microscopic punctured wounds caused by the sharpness of the crystals; and sensitization dermatitis — that is the chemical may be dissolved in the fluid from the seborrheic areas and produce inflammation of the skin, most marked in those areas.[2]

Trinitrotoluene causes a marked deep-orange staining of the skin, by which it is readily absorbed. In the summer of 1919, I treated many cases of poisoning contracted in unloading depth bombs, but only a few cases of active dermatitis were observed. One of those affected was seen two years later, when he was operated on for what was thought to be gall-bladder disease but was actually yellow atrophy of the liver. Trinitrotoluene causes a blue hue on the gums similar to the blue line caused by lead poisoning. This disappears after removal from exposure.[3] A few hours' exposure on a hot day often results in cyanosis, with marked blueness of the lips. Dinitrophenol causes not only local but also systemic disturbances. Explosives, especially those containing potassium dichromate, also affect the mucous membranes, causing perforation of the nasal septum. Nickel and chrome plating are frequent causes of dermatitis. Substitutes are being used in every plant; for example, benzol is now being employed for toluol and may result in blood dyscrasias and cutaneous hemorrhages. Hydrofluoric acid, mercury and magnesium are widely used and all affect the skin.

In airplane manufacture, the agents responsible are: all the halogens; the fish oil, covering the duraluminum, which is laden with bacteria; volatile substances from the paint shop, thinners, dope, special formulas, fumes, varnishes, solvents, gasoline, dryers, asphalts, dyes and oils; and cutting oils in the machine shops (neutral petroleum with free sulfuric acid). Contact with acids, iron oxide, emery, rottenstone and chrome polishing agents may be injurious.[4] Chromic acid compounds cause punched-out ulcerations of the skin and nose, as well as eczema. All the above apply as well to the shipbuilding industry.

In military life, the possibilities for cutaneous diseases are similar to those of industrial and civilian life, varying from sensitivity to clothing and baker's eczema from cinnamon powder or the

persulfates to burns from explosives and gases and infections regarded in civil life as trivial and belonging to the unwashed. However, in military encampments, especially on active service, these diseases become of major consequence, owing to close crowding of men and lack of suitable bathing facilities. Pediculosis vestimentorum capitis and pubis, rarely seen in private practice, infested the personnel of entire camps during the last war, causing great discomfort and severe secondary infections, and were carriers of disease. It is difficult to guard against the single soldier who unknowingly introduces this parasite into a camp. Scabies also caused the loss of many man-days, through failure to recognize the disease and through overtreatment. Scabies alone may be only disconcerting and not disabling, but when combined with sepsis it causes real disability. Every persistent or widespread furunculosis should be regarded as having scabies as a background until such a basis is disproved. Ecthyma and severe pyodermia may result from parasitic infestation.

Fungus infections of the skin are already becoming a problem in the various camps. Before the last war, such infections were endemic in various parts of the tropics. Hence, it was brought by seasoned veterans to training camps, and widespread infection occurred. In turn, these men on discharge brought it to their various communities. The original foci appear harmless: an area of maceration between the toes, especially in the fourth interspace, a few deep-seated vesicles on the central arch of the foot, a yellowish hyperkeratosis under the anterior arch, or a thickened toenail plate may be overlooked or passed over by the examining medical officer. One such focus may infect a whole company. This focus, after a long march or days of sleeping with shoes on, may assume alarming proportions, resulting in the so-called "trench feet" of the last war, or a lymphangitis, an encircling erysipeloid eruption of the lower legs, a phlebitis, inguinal adenitis or even a cellulitis, with resulting serious illness. The fissures caused by fungus infections are frequently portals of entry for streptococci and staphylococci. In this war, world wide as it is, medical officers should be cognizant of fungus infections. At least, they should know those endemic to the neighborhood of their camp, a necessity made evident by the recent outbreak of coccidiosis at a camp in California.[5]

Explosives and Gases

Medical officers and personnel are being trained to cope with the more obvious products of modern warfare, the dangers of explosives and gases. These men are equipped and have adequate protection against gases known to exist, and are therefore ready for any such onslaught; civilian physicians and populations should likewise be informed, trained and equipped. Bit by bit, we are losing our sense of security derived from distance, but this distance still seems to nourish the hope that it would not be worth while to transport wartime gases so far. Why not? They certainly would be sure and as effectual as incendiary bombs. Such an unfamiliar danger could cause great terror and panic. A hit is not necessary — dropping the gas in the vicinity of a plant is enough to disrupt its production at least temporarily. Its effectiveness was demonstrated in the last war. Why have they not been used in this war? It cannot be because of previous treaties, all of which have been flouted; it cannot be because of fear of retaliation, for no such fear on the part of the aggressor has as yet been apparent; it cannot be because of solicitude for the civilian populations, for the bombing of Rotterdam and Coventry belies any such solicitude. It is probably because the rapidity of action to date would make the most valuable gases, the persistent ones, more dangerous to the user than to the enemy; or perhaps up to date the aggressors have not felt the need of these gases and are holding them in reserve in case of necessity. Perhaps the element of surprise, which seems essential in this conflict, would be lacking, for unless the aggressors have developed a new gas they believe that the civilian and military populations are prepared. The time may come when necessity or opportunity or a new discovery will determine their use. I believe that warfare gases will be used on the civilian populations rather than the military, and it consequently behooves physicians to keep informed of progress in this field.

Although many thousands of gases were investigated up to 1918, only a few were found to be effectual, and almost without exception these contained bromine or chlorine atoms. They are effective because of their irritant or destructive action on the skin and mucous membranes. The five most deadly ones are mustard gas (dichlorethyl sulfide), lewisite (vinylchlorazene), phosgene, chloropicrin and bromobenzyl cyanide. Lewisite was not used in the last war. Most of the gases are liquids that, when dispensed as droplets, persist for a long time. Mustard gas, which was the most widely used and was the most effectual, causing the greatest damage to the skin, penetrates paint, woodwork, porous material, shellac, paper, wax, leather and ordinary clothing. Oilskin clothing and synthetic rubber are penetrated slowly.[6] It belongs to the vesicant group — that is, those that cause vesiculation or blistering of the skin. They all present a similar clinical picture — erythema in the folds of the skin, most marked about the geni-

tals, neck and axillas, vesicles and bullae appearing rapidly. The depth of penetration varies. Persons differ in their sensitivities and reactions to these gases, so that the lesions may range from simple vesiculation to necroses, depending also on the concentration and period of exposure.

Mustard gas and lewisite are the most important vesicants. The former causes edema of the lungs, pseudomembranous pharyngitis and destruction of cells and tissue. In civilian populations, chemical warfare is more complicated because of the different ages of the population, and on account of various diseases. Early symptoms may be sneezing only; gradually, the throat becomes irritated. In about twelve hours, there is a free discharge of mucus from the nose. At the same time, the early cutaneous lesions appear. The milder burns are more frequent and insidious, and appear slowly. They are erythematous plaques resembling sunburn. After a while, the central portion desquamates, leaving a slight weeping surface, without any actual blister formation. The earliest lesions are likely to be severe, and are followed by erythematous patches covered with large bullae containing a serous or seropurulent fluid. They occur on the flexor surfaces, particularly about the scrotum, and are frequently replaced by small pustules surrounding the hairs. The pain is commonly severe. Occasionally, necrosis occurs. Itching replaces the pain and may remain indefinitely, being continued by a traumatic neurosis. I have three veterans under treatment who, since 1918, have had pruritus due to mustard-gas burns. Lewisite acts more quickly on the human skin. The latent period is only fifteen to thirty minutes. Its minimum irritant concentration is below its perceptibility through odor. It is apparently absorbed through the skin, causing systemic poisoning. At first, there is a slight burning sensation. After thirty minutes, erythema of the skin occurs and increases gradually. After three hours, areas from 12 to 15 cm. are affected. After thirteen hours, numerous blisters, of cherry size and smaller, appear and gradually coalesce in one large blister,[7] which later becomes surrounded by an erythematous halo. These areas may slough down to the muscles, and unlike mustard gas, which usually leaves only atrophy, lewisite may result in deep scarring. The pain accompanying these burns is usually severe. I have already seen one patient with a burn from lewisite. He was an instructor who demonstrated the art of sniffing once too often. He showed marked erythema and desquamation of the skin of the face and neck. Death may occur from both gases, but more frequently results from lewisite.

Burns from caustics have been seen particularly from sodium hydroxide, which causes lesions varying from a few vesicles to deep ulcerations (Fig. 5). With the maintenance of military secrets, it

FIGURE 5. *Punched-out Lesions from Caustics.*

is impossible for the outside physician to know exactly what the exposure is, but careful scrutiny of the skin — especially of the hands for yellow, brown or gray stains, or a bluish appearance of the mucous membranes, or punctate hemorrhages — will lead to the suspicion of systemic poisoning.

PREVENTION

Most occupational dermatoses can be prevented. Hazards must be detected and eliminated. Mechanical devices should replace manual contacts, and when this is impossible, gloves should be worn; when gloves cannot be worn, protective liquids or creams should be tried. Such creams must be selected with care, for they can cause eruptions. Workers exposed to irritants and oils should be taught the value of cleanliness, not only of themselves but also of their benches and machines, and of removing the grime before meals and of applying an emollient at bedtime, and again before starting work. Periodic instruction to foremen is highly recommended; they should be taught the need of careful supervision of their men and women workers, so as to prevent them from discarding gloves and from using dangerous short cuts to speed up piecework, such as dumping articles in bowls of acids or caustics and removing them by hand instead of by tool, as well as using fingers, cloths or sponges in place of brushes.[8] They should watch for signs of fatigue or carelessness or recklessness, all of which are the chief causes of many injuries. Hazardous processes should be safeguarded whenever possible. No new substances should be introduced until their possible effects on the workers have been thoroughly studied. In

many cases, dangerous chemicals are preferred to less irritating ones because of their lower cost—for example, the use of benzene instead of naphtha; moreover, since many chemicals are not now available, substitutes must be used. No worker should be compelled or permitted to keep his hands wet with any solution for hours at a time, for few skins can stand constant wetting. Such jobs should be alternated during the day with those in which the hands must be kept dry. Young workers, notably those under twenty years of age, should not be employed in hazardous occupations, such as the manufacture of explosives.

In military medicine, the best method of prevention is the instruction of medical officers in the early recognition of dermatoses, careful inspection of newcomers to camp and segregation of those with contagious cutaneous disease. Treatment of these diseases should be by recognized, proved therapy. Experimental work should not be tried at military camps, but at home by those who are unable to enter active service. Perforce, much will be learned, by accident or by mistakes. It is already known not only that the sulfanilamides are neither bactericidal nor fungicidal but also that both organisms will grow in their presence. They must be used with care, especially in persons who have industrial exposure to magnesium or sulfate. I have seen one case of a generalized dermatitis following the ingestion of two tablets of sulfathiazole in a worker who had such an exposure. Whether this was due to a peculiar idiosyncrasy of the patient who had never taken any medication or to this exposure is impossible to state. Their use locally should be limited to fresh preparations or those sterilized.

Physicians must be alert to the dangers of epidemics, especially in communities near military encampments. They should be experts in first aid and should have a fundamental knowledge of wartime gases. The civilian population should be taught what to do in such raids. Minutes are important. With lewisite, preventive treatment must be started immediately. By the time a patient reaches a hospital or a decontamination station, it is too late. Civilians must be taught sensible methods, and just as every household is now informed what to do in case of an incendiary bomb, so too each one should be taught the essential measures to be taken immediately in case of a gas attack, for their own protection and for the relief of others. All these directions may be learned from several pamphlets, the most recent and thorough being the one published by the United States Office of Civilian Defense and entitled *Protection Against Gas*. A can of kerosene, a bottle of hydrogen peroxide and a package of bleaching powder or pail of soapy water, timely and properly used, may save lives.

* * *

A knowledge of dermatology, always important in civilian life, is most essential in industrial and military organizations. Dermatologists have already demonstrated their value in industry. They promoted the Council on Industrial Health of the American Medical Association, pointed out many serious hazards, and suggested methods to prevent them and ways to relieve those affected. The last war taught the value of specialization. In military life, dermatologists should be assigned to their specialty and given an opportunity to teach other medical officers the prevention, diagnosis and treatment of cutaneous diseases. It may not sound very serious to mistake a case of dermatitis due to a hypersensitivity to clothing for a case of scabies or vice versa, but wrong treatment might be serious to the victim as well as to the service when a few days' disability is extended to weeks or months, with great loss of manpower. The training of dermatologists in syphilology should place them in charge of the control and the diagnosis and treatment of this disease. The danger of new and unknown poisons and diseases, against which there is no defense and about which experience alone can teach one, will be ever present in this conflict, so that a constant need of medical literature on these subjects will arise. Hence, the value of continuing medical publications and medical meetings for the dissemination of recent experiences and results is evident. The dangers of the present conflict should be a challenge to the entire medical profession, who should not fear these dangers but should be prepared and willing to share any exposure, hardship or risk to attain the ultimate objective—victory.

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References

1. Hilliard, C. M. Health of the worker and his family. *New Eng. J. Med.* **225**:1033, 1941.
2. Silver, A. L. L. The treatment and prevention of industrial diseases in filling factories. *J. Roy. Army M. Corps* **71**:87-96, 1938.
3. Schwartz, L. Cited by Lounsberry.[4]
4. Lounsberry, C. R. Occupational dermatoses in the aircraft industry. *California & West. Med.* **51**:309-313, 1939.
5. Shelton, R. N. A survey of coccidioidomycosis at Camp Roberts, California. *J. A. M. A.* **118**:1186-1190, 1942.
6. Goldman, L., and Cullen, G. E. The vesicant chemical warfare agents. *Arch. Dermat. & Syph.* **42**:123-136, 1940.
7. Wachtel, C. *Chemical Warfare*. 312 pp. Brooklyn: Chemical Publishing Co., 1941.
8. Downing, J. G. Cutaneous eruptions among industrial workers. *Arch. Dermat. & Syph.* **39**:12-32, 1939.

RETENTION OF A RUBBER TUBE IN THE BILIARY TRACT

Report of a Case

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THE purpose of this article is to report a case in which a rubber tube was retained for four and a half years in a choledochoduodenostomy communication without any untoward symptoms and with, eventually, the expulsion of the tube and complete restoration of the biliary-duodenal continuity.

CASE REPORT

A 36-year-old married woman was admitted to the Malden Hospital on January 17, 1937, with the complaint of jaundice, itching of the skin and pain in the upper abdomen for the previous 3½ years. There had been marked increase of jaundice, itching and pain for 6 weeks prior to admission. The stools, which heretofore had been clay colored during the attacks and light yellow during the interim, had been clay colored for the preceding 6 weeks.

The patient dated her present complaints to about a year following her first operation, which was performed elsewhere in May, 1932. The gall bladder containing stones was removed, after which there was drainage of bile for 5 months. The wound then healed, and the patient remained in good health for several months. In June, 1933, approximately a year after the cholecystectomy, the upper abdominal pain, jaundice and itching of the skin occurred. The jaundice lasted 2 or 3 weeks and was accompanied by intense pain and itchiness, with clay-colored stools and dark-brown urine. Following each attack, the patient was symptom free for a similar period. To relieve her condition, a second operation was performed on October 31, 1933, at another hospital, and a side-to-side choledochoduodenostomy was performed for complete occlusion of the common duct in the second part of its course.

After this operation, the patient was symptom free for about 8 months, when the symptoms of which she previously complained gradually reappeared. At first moderate, the intermittent attacks of pain, jaundice and itching became quite frequent and prolonged on each attack. For 6 weeks before admission, the patient had been quite ill, having had no relief whatsoever. Her skin had become a deeper bronze, and the epigastric discomfort and pain with chills became persistent. At times, the pain, chills, fever and insomnia were so marked and vomiting so severe that the patient had to remain in bed for days. Finally, she sought relief because her condition became progressively worse.

Physical examination revealed a very thin and moderately emaciated woman, markedly jaundiced. The scleras and skin had a deeply bronzed appearance. There were no enlarged lymph nodes. The heart and lungs were essentially normal. The blood pressure was 112/64, the pulse 66, and the respirations 18. The temperature was 98.6°F. The abdomen was rather soft and scaphoid, and revealed a firmly healed incision, about 13 cm. long, below and parallel to the right costal arch. Palpation elicited a definite area of tenderness, moderate muscular spasm and rigidity above and to the right of the umbilicus. No masses were felt. The liver was very much enlarged and easily palpable, and extended almost to the brim of the right pelvis; the spleen was not enlarged. Many streaked abrasions were present on the arms, legs and body, denoting continuous scratching. Her weight was 121 pounds — 39 pounds below her usual weight.

On admission to the hospital, the value for hemoglobin was 80 per cent (Sahli); the red-cell count was 3,500,000, and the white-cell count 9000. The icteric index was 100, as compared with the normal value of 5, and the serum bilirubin (van den Bergh reaction) was 82.5 mg. per 100 cc. The bleeding time was 4½ minutes; clotting began in 5 minutes and was complete in 6 minutes. The blood

FIGURE 1.

This film, taken thirteen weeks after operation, shows extensive filling of the bile ducts and liver radicles immediately following the injection of Hippuran solution through the T tube. Small amounts of the opaque material are visible in the duodenum.

Wassermann reaction was negative. Urine specimens contained a large amount of bile, and the stools were clay colored. A flat x-ray plate of the abdomen revealed no visible calculus in the gall ducts, but the liver was moderately enlarged.

In view of the history and the physical and laboratory findings, a diagnosis of an obstructed choledochoduodenostomy stoma was made, and operation was advised to restore the flow of bile into the intestinal tract. Hypodermoclysis of 500 cc. of 5 per cent glucose twice daily and 15 gr. (1 gm.) of calcium chloride daily were given for 3 days, and on the day prior to operation 475 cc. of citrated blood was given.

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The operation was performed under nitrous oxide, oxygen and ether on January 21, 1937. A Mayo-Robson incision through skin and fascia was carried out; the rectus muscle was divided longitudinally, and the peritoneum opened. Omental adhesions were released from the parietal peritoneum and also from the anterior surface

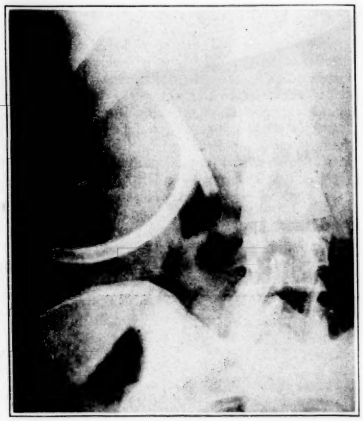

FIGURE 2.

This film, taken five hours after the injection of Hippuran solution, shows complete disappearance of the opaque material. None of the opaque solution is visible in the intestinal tract. There is air in the duodenum.

of the liver. The liver was greatly enlarged, especially the right lobe, which extended almost to the pelvic brim. Furthermore, omentum adherent to the gall-bladder fossa was released without difficulty, part of the inferior surface of the liver and the first portion of duodenum being brought clearly into view. A bluish discoloration of the biliary duct was readily recognized, and it was evident that the hepatic duct extending 3 cm. downward from the liver was firmly attached at a right angle with the second portion of duodenum. The hepatic duct was then aspirated with a needle and syringe, and clear bile obtained. Further inspection revealed that the hepatic duct was under tension and its walls moderately thick, and that throughout its length — from the inferior surface of the liver to the junction of the duodenum — its width was about 9 mm. Two linen traction sutures were inserted in the wall of the hepatic duct (about 3 mm. apart), and the duct opened through a longitudinal incision. Bile flowed freely, and many small inspissated particles of bile were extracted. Exploration of the duct with a probe, however, revealed that the biliary duodenal stoma was occluded and did not allow the probe to enter the duodenum. The incision of the hepatic duct was then extended through the stoma and into the duodenum, the stoma being thus enlarged. A T tube was then inserted, connecting the hepatic duct with the duodenum. Several interrupted linen sutures were used to approximate the incision and reinforced with a portion of omentum sutured with No. 00 plain catgut. A cigarette drain was inserted in Morrison's pouch, and the T tube and drain brought out through a stab wound lateral to the incision. The abdomen was closed in layers, No. 2 chromic catgut being used for the peritoneum and fascia; four stay sutures of silkworm gut were inserted, and the skin was sutured with silkworm gut interruptedly. The operation lasted 85 minutes, and the patient was in excellent condition at its termination. She was given intravenous glucose, and the T tube connected with tubing was brought into a bottle at the side of the bed.

The postoperative course was uneventful. There was slight elevation of temperature for the first 3 days, but the general condition was greatly improved. The appetite was good, the skin took on a lighter hue, and scratching gradually subsided to a minimum degree; the feces, which had taken on a yellowish color 24 hours after the operation, gradually became normal brown in appearance, and the icteric index dropped from 100 to 60. Biliary drainage averaged 100 cc. per day, although the bile was quite viscid at first and very small in quantity; however, with

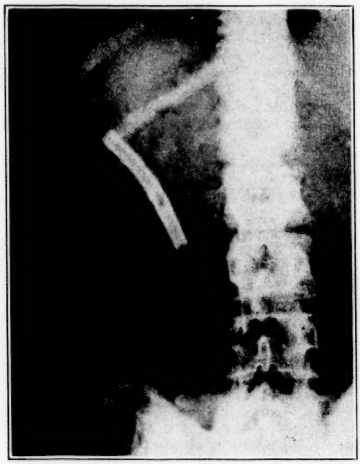

FIGURE 3.

This film shows the horizontal part of the T tube in the biliary duodenal channel; the tube broke at time of removal sixteen weeks after operation. The oval area in the tube indicates where the long arm was cemented.

the administration of bile salts and iron, a definite improvement was noted in the specific gravity of the bile, and the patient regained considerable strength. She was discharged from the hospital on February 6, 16 days after operation. The total hospital stay was 20 days.

At home, temporary clamping of the T tube did not cause any discomfort, and 6 weeks after the operation the tube was finally left clamped at all times. This caused no pain or discomfort, and the stools remained brown. The patient ate about everything without any complaints. On April 21 (13 weeks postoperatively), roentgenographic and fluoroscopic examinations were made by Dr. Max Ritvo (Figs. 1 and 2).

On May 20 (16 weeks postoperatively), under nitrous oxide and oxygen anesthesia, the T tube was removed. In the process of extraction, the long arm of the tube broke off, leaving the T portion of tube in situ (Fig. 3). For a week, bile drained through the skin opening but gradually decreased, and the wound closed 14 days after this mishap. There was no change in the patient's condition, and her stools continued to be brown. She had no digestive disturbances and regained much of her former weight.

FIGURE 4.

This film, taken four and a half years later, shows absence of tube and depicts liver radicles and duodenum filled with air.

X-ray films were then taken at intervals from 6 months to 1 year, and on each occasion the tube was found to be snugly in its original position. On February 7, 1942, however, the tube was no longer present (Fig. 4), and it was evident that the patient had unconsciously expelled the tube. From this, it can safely be said that the tube was in its original place for at least 4½ years. Throughout this period, the patient experienced no untoward symptoms, and for the last year had been working in a laundry.

On her last visit, February 7, she was feeling very well and had no complaints except a sense of fullness in the upper abdomen, especially after a large meal. The stools had been brown, and her weight had remained at 140 pounds. There was no jaundice, the abdominal incision was well healed, and the liver was smooth, 3 fingerbreadths below the costal margin.

DISCUSSION

This case illustrates several interesting points. The first, of course, concerns the reliability of the sidetracking operation of choledochoduodenostomy in stricture of the common duct.[1-4] That it is not always a successful procedure is well substantiated by men of experience.[5, 6] Others, dissatisfied with the unsatisfactory results, have resorted to intubation.[7, 8] The varied opinions clearly indicate that these difficult cases are sometimes prone to failure, regardless of procedure. Unquestionably, a prognosis is most difficult to make in such cases.[9]

The second point of interest is that in this case the horizontal part of the T tube broke off at the time of removal, sixteen weeks after its insertion to relieve obstruction following choledochoduodenostomy. Whether this incident was a piece of good fortune is difficult to say. The fact remains that the broken-off horizontal part of the tube remained within the biliary duodenal channel for four and a half years without causing the usual complications of cholangitis and obstruction. Even after the expulsion of the tube, the biliary duodenal communication continued to function very well, although, of course, it is rather soon to venture any prognosis.

This incident does not appear to be the first to be reported. Fedoroff[10] had a similar experience. In his case, the horizontal portion, which broke off at time of removal, was 6 cm. long, and remained within the biliary channel also for four and a half years. When removed, it was found to be covered with *Gallensand*. In Bowen's[11] case, it was not the horizontal portion that broke off but the end of a No. 20 Fr. catheter that had been inserted in the common duct; it was 3.8 cm. long and remained in situ for four years.

Possibly the construction of the tubes has some connection with these unusual accidents. Bowen[11] insists on the use of a well-made new tube and states that the tubing used should be easily visualized roentgenographically. In the case presented above, the long portion of the T tube was cemented to the short, and for this reason I am fairly well convinced that the construction and, perhaps, the age of the tube were in some measure responsible. Since this mishap, whenever the occasion arose to use a T tube, I have made sure that it was a single piece and never used before.

The final point of interest is that unintentional intubation resulted and the tube was retained for four and a half years without the usual complications. Rubber tubes within the biliary channel have been known to be retained for prolonged periods, even up to many years. The longest recorded period is twelve years[12]; the interior of the tube became encrusted with bile-pigment debris, which obstructed it. Lahey[13] cites a case in which he removed from the common duct a tube that had functioned well for seven years and replaced it with another. At the time of his report, the second tube was still functioning satisfactorily. Behrend[14] reports a case in which a straight tube

was inserted and the common duct repaired over it for reconstruction of the duct. When the tube was removed six years later, it was encrusted with bile salts. Hunt[15] likewise reports a case in which a tube remained in the hepaticoduodenostomy stoma five months and ten days. He also mentions the fact that he saw an autopsy specimen in which such a tube had remained in place for eight years. Colp,[7] in presenting his intubation method, refers to a case reported by Judd in which the tube remained in place for four years before it caused cholangitis.

I am fully aware that there are just as many interesting cases in the literature that I was unable to ferret for the simple reason that most of the cases in which a rubber tube was retained for a prolonged period have been solitary experiences and casually mentioned by the authors.

Summary

Choledochoduodenostomy is occasionally prone to failure and fraught with serious complications and experiences.

A case is presented in which the horizontal part of a T tube broke off at the time of removal and became an indwelling rubber tube. It was retained within the biliary duodenal anastomosis without any complications for four and a half years.

In such operations, the use of a new T tube, constructed in one single piece, is recommended.

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References

1. Nygaard, K. K., and Sheiden, C. H. Strictures of the bile duct: results in fifty-one operative cases. *Proc. Staff Meet., Mayo Clin.* **12**:25-30, 1937.
2. Walters, W. Complete stricture of the common and hepatic ducts: treated by transplantation of external biliary fistula into the stomach or duodenum: report of five cases. *J. A. M. A.* **96**:1121-1124, 1931.
3. Judd, E. S., and Parker, B. R. Biliary intestinal anastomosis for obstructive jaundice: analysis of one hundred and thirty-seven consecutive cases. *Arch. Surg.* **17**:1-17, 1928.
4. Strauss, A. A., Strauss, S. F., Crawford, R. A., and Strauss, H. A. Chronic biliary stasis: treatment by choledochoduodenostomy and gastroenterostomy. *J. A. M. A.* **101**:1365-1371, 1933.
5. Schrager, V. L., Ivy, A. C., and Morgan, I. E. A method for the plastic reconstruction of the common bile duct: an experimental study. *Surg., Gynec. & Obst.* **54**:613-619, 1932.
6. Douglas, John. Strictures and operative injuries of the bile ducts. *Ann. Surg.* **84**:392-403, 1926.
7. Colp, R. Biliary duodenal intubation. *Surg., Gynec. & Obst.* **63**:157-162, 1936.
8. Beye, H. L. Conditions necessitating surgery following cholecystectomy: an analysis of sixty-six cases and a discussion of certain technical problems concerned in removal of the gall bladder and in operations upon the common bile duct. *Surg., Gynec. & Obst.* **62**:191-202, 1936.
9. Deaver, J. B., and Burden, V. G. The common bile duct. *Surg., Gynec. & Obst.* **53**:662-666, 1931.
10. Fedoroff, S. P. Drainrohr im Choledochus 5½ Jahre lang. *Zentralbl. f. Chir.* **57**:657-662, 1930.
11. Bowen, F. H. Foreign bodies in the biliary tract: report of a case with a tabular review of the literature. *Arch. Surg.* **43**:458-461, 1941.
12. Queries and Minor Notes. Duration of retention of rubber tube in bile duct. *J. A. M. A.* **111**:1040, 1938.
13. Labey, F. H. Strictures of the common and hepatic ducts. *Ann. Surg.* **105**:765-790, 1937.
14. Behrend, M. The T tube in surgery of the common duct. *S. Clin. North America* 1839-1847, 1940.
15. Hunt, V. C. Surgical clinics: second day: the common duct in relation to surgical conditions of the biliary tract. *J. Iowa M. Soc.* 473-477, 1934.

THE CLINICAL APPLICATION OF GASTROSCOPY*

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BOSTON

THE semiflexible gastroscope was introduced ten years ago; as a result, gastroscopy became a relatively simple technical procedure. In the hands of an experienced examiner, it has proved to be a safe method of visualization of the gastric mucosa. Illustrating its increasing popularity is the fact that about four hundred and fifty semiflexible gastroscopes are now in use in this country. Most of these are of German make, but an American-made gastroscope has recently been introduced, and its satisfactory performance is vouched for by Schindler and others who have used it.

The proper evaluation of a new method of diagnosis at best takes time and the collective efforts of the medical profession. Such efforts include those of the enthusiastic pioneer and specialist, who can compare the objective findings by a new technic with those of the older and well-established methods. However, the clinical status of a new procedure is ultimately decided by the rank and file of the profession, who learn from experience whether this method helps them in their medical practice. These observations furnish the plan that has governed our investigation of the clinical value of gastroscopy. This investigation was incident to the search for methods to increase our diagnostic acumen and persistently carried on in the Gastrointestinal Clinic of the Out-Patient Department of the Boston City Hospital. So far, we have performed over 1500 gastroscopies on patients for whose clinical diagnosis we were responsible. Thus, we have been familiar with their entire clinical record, including x-ray and other special findings. It is on the basis of these observations that we present our conclusions concerning the clinical value of gastroscopy.

In the evaluation of gastroscopy as a method for objectively demonstrating intragastric disease,

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it is well to compare its relation to roentgenology of the stomach. The two diagnostic procedures are complementary, and are neither competitive nor antagonistic. With the judicious use of both, the error in gastric diagnosis can be reduced considerably.

In our clinical study of the patient, we first obtain a careful history and make a complete physical examination and indicated laboratory tests. Barium x-ray observations are next made for general orientation of digestive-tract disturbances and as an aid in excluding conditions that may contraindicate gastroscopy. After these examinations, the use of gastroscopy is given consideration for two reasons: in the presence of a gastric lesion demonstrated by x-ray study, direct inspection through the gastroscope may furnish evidence enabling a much more exact differential diagnosis; and gastroscopic inspection may disclose an intragastric lesion that the x-ray examination failed to demonstrate. These practical applications of gastroscopy will be clarified by a description of its use in four intragastric lesions. Lack of space precludes the discussion of fissure, superficial ulceration, hemorrhagic areas, diverticulum, petechiae, sarcoma, lymphoblastoma and gumma, or of observations on postoperative cases.

Of the four important intragastric lesions, chronic gastritis is by far the most frequent. The incidence of chronic gastritis is given as 42 to 44 per cent of all cases examined (Schindler,[1] Carey[2] and McClure, Sweetsir and Jankelson[3]). In a previous communication,[4] however, we have emphasized that, in approximately 50 per cent of the cases, the gastritis is secondary either to another intragastric or to extragastric disease. By making this statement, we do not choose to commit ourselves regarding whether a chronic gastritis precedes an ulcer or cancer of the stomach or is the result of a pre-existing lesion. This is another chapter whose discussion would lead us far astray from the original purpose of this contribution. What we imply with the term secondary chronic gastritis is the presence of other lesions within the stomach or elsewhere in the body, frequently associated with inflammatory or noninflammatory changes in the gastric mucosa.

Chronic gastritis may be hypertrophic, atrophic, superficial or mixed, and its distribution may be localized or diffuse. In its clinical course, it may simulate a peptic ulcer or be mistakenly diagnosed as a gastric neurosis. Although the clinical diagnosis of chronic gastritis may be suspected, it cannot be made with certainty, and the different types cannot be recognized. The changes in gastric secretions are not diagnostic, and the demonstration of an excess of mucus in the gastric contents, as well as the presence of leukocytes, is not conclusive evidence. Generally, the clinical laboratory gives indefinite information in this disease. The atrophic and superficial types cannot be demonstrated by either fluoroscopy or x-ray study. The frequently seen large rugae within the stomach are not necessarily due to hypertrophic gastritis (Berg[5] and Ansprenger and Kirklin[6]). Thus, the roentgen rays are not diagnostic. The only clinical method for ascertaining the presence, type and distribution of gastritis is gastroscopy. The use of gastroscopy for this purpose has impressed us with the fact that minor variations in the appearance of the gastric mucosa as seen through the gastroscope should not be emphasized and that only gross variations from normal are of diagnostic significance. More recently, Swalm and Morrison,[7] in a gastroscopic study accompanied by biopsies and microscopic examinations of specimens of the gastric mucosa, came to the same conclusions. Their microscopic studies showed a high percentage of verification of the gross picture of chronic gastritis in the cases in which the changes were marked, whereas in the minor variations the error was much greater. Thus, one may say that the diagnosis of chronic gastritis is largely made on the basis of gastroscopic findings. Very similarly, lesions that often accompany a chronic gastritis, such as fissures, submucous hemorrhages, petechiae and small inflammatory polyps, can be recognized clinically only with the aid of a gastroscope. Without it, these lesions may be suspected, but a definite diagnosis is impossible.

The second lesion, peptic ulcer, may be diagnosed clinically if a typical history is obtained. However, an ulcerative gastritis, an ulcerating carcinoma of the stomach or multiple fissures of the stomach may mimic a peptic ulcer to an extent that a differentiation becomes clinically impossible. Moreover, the localization of an ulcer is impossible on the basis of history, physical examination or clinical laboratory data. We need not discuss the necessity of differentiating a gastric ulcer from a duodenal one. Suffice it to say that the former is potentially a cancer, whereas the latter almost never becomes carcinomatous. Hence, every clinical diagnosis of peptic ulcer or a serious suspicion of one requires verification and localization with the aid of a barium meal and fluoroscopic and roentgenographic studies. Thus, the majority of ulcers are located, their sizes and depths determined, their progress evaluated, most of the complications discovered, and the indications for surgery confirmed. However, many ulcers within the stomach are so situated or so superficial that

they cannot be visualized by roentgen rays; frequently, these can be seen by gastroscopy. Furthermore, one cannot determine whether some ulcerative lesions are benign or malignant on roentgenologic evidence, even if the examination is repeated at frequent intervals. However, the gastroscope frequently contributes information that permits the proper classification of such lesions.[8] In the gastroscopic examination, not only the appearance of the ulcer but also the character of the gastric mucosa of the rest of the stomach may give evidence of the character of the lesion. For example, in the vast majority of benign ulcers, the gastric mucosa appears normal or hypertrophic changes are present, in carcinoma, almost invariably, the entire gastric mucosa is atrophic, whereas in an ulcer that has undergone malignant degeneration only the adjoining mucosa is atrophic.[9]

Although healing of an ulcer can be demonstrated by roentgen rays, at times this evidence is misleading because the niche may appear smaller or less penetrating as a result of exudate accumulated on its base and not due to repair. Such a condition is demonstrable by gastroscopy, which in our experience has proved itself superior to roentgen rays in following the progress of the healing. With the aid of preoperative and postoperative gastroscopy, one makes observations pointing to a major factor in the prevention of the late postoperative gastritis that may accompany peptic ulcer. Our studies indicate that the ultimate results in ulcer surgery are better if the operation is performed at a time when an accompanying gastritis is properly controlled than when it is active. On the other hand, a gastric ulcer not visualized on gastroscopy may occasionally be demonstrated by radiography; therefore, the inability to see an ulcer by gastroscopy does not exclude its presence.[10] Thus, the use of both methods is strongly advocated to reduce the error of diagnosis.

The diagnosis of the third lesion, carcinoma, is usually easy in advanced cases. The appearance of the patient, rapid loss of weight, epigastric distress and anorexia make one suspect it whether or not a palpable mass is present in the epigastrium. Almost invariably, the tumor mass can be located and its extent determined roentgenologically. In other words, only very rarely do the roentgen rays fail to visualize an irregular, moth-eaten deformity of the stomach. However, in a few cases of extensive carcinoma of the stomach, usually involving the posterior wall or the cardiac end, the routine x-ray examination is either inconclusive or negative. In such cases, gastroscopy is indicated and often solves the diagnostic problem. In our experience, gastroscopy is of great help in determining the extent of involvement of the stomach. On the other hand, gastroscopy gives no information concerning regional lymph-node involvement or distal metastases and therefore does not in itself settle the question of operability. Obviously, resectability can be determined clinically only by employment of all methods of examination at one's disposal, including peritoneoscopy.

The early diagnosis of cancer of the stomach is often difficult. The condition is only suspected clinically, and because of the value of an early diagnosis all methods of clinical investigation should be utilized. Roentgen rays and gastroscopy are the two major methods of arriving at an objective diagnosis. If the lesion involves either gastric orifice, the x-ray evidence is usually conclusive. But when the early carcinomatous lesion is away from the cardiac or pyloric orifice, gastroscopy, at least in our experience, frequently becomes essential in the making of a definite diagnosis.

The fourth group of lesions, benign tumors, usually comprise polyps and leiomyomas. The former originate from the mucosa, protrude into the cavity of the stomach, vary in size from a pea to a walnut, and may be single or multiple. A generalized polyposis of the stomach also occurs. The seriousness of polyps lies in the fact that they may ulcerate and bleed and that more than 40 per cent undergo malignant degeneration. Although the larger ones can be demonstrated roentgenologically, the smaller ones may escape detection; thus the radiographic demonstration of gastric polyps is uncertain. Gastroscopically, however, the polyps can be seen, their number determined, their size estimated, ulcerations or bleeding points demonstrated and areas of early malignant degeneration often observed.

The leiomyomas spring from the muscle layers of the stomach, usually in the cardiac end, and may appear as submucosal or subserosal tumors, covered with normal mucous membrane. They too may bleed when they ulcerate and show a tendency to sarcomatous degeneration. Since these lesions are covered by normal mucosa, their character cannot be determined by gastroscopy. Particularly, the subserosal ones, cannot be seen through the gastroscope. The roentgen rays, however, most frequently give information of their location, size and character. These observations emphasize the desirability of both methods in gastric diagnosis.

Whereas other methods, such as roentgenology, give objective information not only about the stomach but also about the adjacent organs, such as the esophagus and duodenum, the scope of gastroscopy is much more limited. Only the mucosa of the stomach can be seen by this method of examination. Moreover, even within the gastric cav-

ity, certain areas may not be visualized and thus a localized lesion may be overlooked on gastroscopy. On the other hand, not only is a lesion located through this instrument but also much information about its character, type, extent and distribution is obtained. Primarily, the gastroscope gives information about the gross pathologic changes.

Gastroscopy has limitations. In carcinoma, the walls of the stomach may become rigidly infiltrated and lose their distensibility, visualization through the gastroscope being prevented. Although lesions involving the entire gastric mucosa, such as diffuse gastritis, lymphoblastoma and polyposis, can hardly be overlooked on gastroscopy, localized lesions — an ulcer or a single polyp — can be missed on a single examination. The dependability of this procedure in the final analysis depends largely on the experience of the examiner. Besides these limitations, there are contraindications to gastroscopy, which should be scrupulously observed, lest unnecessary accidents be encountered and serious injuries produced to the patient. Since we[11] have already listed such contraindications to and limitations of gastroscopy, the reader is referred to this previous publication.

* * *

Roentgenology and gastroscopy are complementary methods of diagnosis, and thus are neither competitive nor antagonistic. In other words, gastroscopy is an additional method of arriving at an anatomic diagnosis in diseases of the stomach. As such, it does not exclude any other form of examination. In our experience, it has proved to be a useful procedure, which in many cases has given evidence not obtained by any other method of examination and in others has confirmed evidence otherwise recognized or suspected. Judiciously used, gastroscopy has enlarged the scope of diagnostic acumen. For example, it has rehabilitated the concept of chronic gastritis. It has proved to be a useful procedure in the diagnosis of gastric ulcer and cancer, in preoperative and postoperative observations of the gastric mucosa and in the differential diagnosis of gastric ulcer and cancer. In our opinion, gastroscopy furnishes objective findings whose clinical significance should be established in relation to the history, physical examination, laboratory findings, gastrointestinal roentgen-ray studies and all other special studies indicated.

References

1. Schindler, R. The incidence of the various types of gastric disease as revealed by gastroscopic study. *Am. J. M. Sc.* **197**:509-516, 1939.
2. Carey, J. B. Gastroscopic observations in chronic gastritis. *Am. J. Digest. Dis.* **7**:160-164, 1940.
3. McClure, C. W., Sweetsir, F. N., and Jankelson, I. R. Chronic gastritis: a gastroscopic and clinical study. *New Eng. J. Med.* **225**:259-263, 1941.
4. McClure, C. W., and Jankelson, I. R. Gastroscopic observations on chronic gastritis. *J. Maine M. A.* **30**:24-27, 1939.
5. Berg, H. H. *Röentgenuntersuchungen am Innenrelief des Verdauungskanals.* 198 pp. Leipzig: G. Thieme, 1930.
6. Ansprenger, A. G., and Kirklin, B. R. The roentgenologic aspects of chronic gastritis. *Am. J. Roentgenol.* **38**:533-541, 1937.
7. Swalm, W. A., and Morrison, L. M. Gastroscopic and histologic studies of the stomach with gastric and extra-gastric disease during life and at autopsy. *Am. J. Digest. Dis.* **8**:391-397, 1941.
8. Schindler, R., and Giere, N. Gastric surgery and gastroscopy: differential diagnosis of benign and malignant lesions; operability of tumors as determined by gastroscopy; early diagnosis of gastric carcinoma; the postoperative stomach. *Arch. Surg.* **35**:712-765, 1937.
9. Orator, V. Beiträge zur Magenpathologie II: zur Pathologie und Genese des Carcinoms und Ulcuscarcinoms des Magens. *Virchows Arch. f. path. Anat.* **265**:202-229, 1925.
10. Jankelson, I. R., and McClure, C. W. Limitations of gastroscopy. *New Eng. J. Med.* **224**:64-67, 1941.
11. *Idem.* Observations on the clinical status of gastroscopy. *New Eng. J. Med.* **219**:917-920, 1938.

MEDICAL PROGRESS

STILBESTROL

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"STILBOESTROL" was the name given by Dodds, Goldberg, Lawson and Robinson[1] in 1938 to the compound, 4:4′-dihydroxystilbene, which they prepared from an extract of the oil of aniseed, and which they discovered to be estrogenic. The diethyl derivative (4:4′-dihydroxy-$\alpha\beta$-diethylstilbene) of this synthesized vegetable hydrocarbon, however, has been found to be so much stronger in estrogenic effect as to make stilbestrol itself clinically unimportant.

GENERAL CONSIDERATIONS

Nature of Drug

The precise Council on Pharmacy and Chemistry of the American Medical Association[2] prefers to limit the name "stilbestrol" to Dodds's original simpler and weaker estrogen (4:4′-dihydroxystilbene) and uses the rather cumbersome descriptive name "diethylstilbestrol" for the more potent derivative now in such general use as practically to exclude the weak mother substance. In this paper, stilbestrol is employed to designate the drug in common use, 4:4′-dihydroxy-$\alpha\beta$-diethylstilbene.

Whether used in tablets, in suspensions or in solutions, or whether given by mouth, by injection or implantation, by way of the vagina or percutaneously, stilbestrol, although it is not a true hormone, appears to have all the clinical estrogenic power of the female sex hormone, estradiol, and its various derivatives.[3–6] Whereas Greene[6] rates 1 mg. of stilbestrol as equivalent to about 20,000 I.U. (international units), or 2 mg., of estrone when both are given parenterally, the same effect is produced by only 0.3 to 0.4 mg. of estradiol benzoate similarly administered. Stoddard and Metzger[7] report that in the human subject 0.42 mg. of estradiol benzoate, injected intramuscularly, is equaled by 0.5 mg. of stilbestrol taken orally.

The distinct advantage of stilbestrol over estradiol, clinically the most potent of the estrogens, is thus evident. Whereas estradiol loses most of its efficacy when taken orally, stilbestrol is almost as potent by mouth as by injection.

Cost

A second decided advantage of stilbestrol over estradiol is its lower cost. On the basis of human assay, the result produced by the intramuscular injection of 0.42 mg. of estradiol benzoate, priced at $1.07, may be duplicated by the oral consumption of 0.5 mg. of stilbestrol, costing less than 2 cents.[7]

Pharmaceutical Forms

The drug is dispensed in small tablets, some of which are enteric coated, as well as in capsules and suppositories. Soon, no doubt, it will be obtainable in solutions or suspensions for percutaneous use, as well as in pellets that can be implanted, since these forms have been successfully employed in clinical experimentation.[8, 9] It is questionable, however, whether these will ever be of great value, since stilbestrol is so effective by mouth.

Dosage

The effective dose of stilbestrol varies considerably because patients have widely different susceptibilities, both to desired and undesired results, and because among the many disorders that it may relieve, some are easily affected, whereas others are very resistant. Since the drug may have unpleasant sequelas, as mentioned below, it is wise to begin treatment with as little as 0.1 mg. by mouth daily. If well tolerated, this may be increased even up to 5 mg. daily, although only occasionally is it necessary to exceed 1 mg., and only rarely, as for the suppression of lactation, is one obliged to give more than 2 mg. daily. When injected, it should be put in muscle, and then only on every third or fourth day; the gluteus medius on alternate sides is very suitable.

Utilization

Stilbestrol is effectively absorbed from both the intestines and the muscles but is not utilized in the same degree from these two sources. On the basis of analyses made on 3 menopausal patients, von Haam et al.[10] estimated that 10 to 30 per cent of a single large dose of injected stilbestrol was ex-

Reprints of articles in this series are not available for distribution, but the articles will be published in book form. The current volume is *Medical Progress: Annual, Vol. III, 1942* (Springfield, Illinois: Charles C Thomas Company, 1942. $5.00).

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creted in the urine and feces during the first ten days after its administration. Zondek[11] reported that one fourth of the amount injected into infantile rats could be recovered from the excreta. He also found that, forty-eight hours after injection, stilbestrol could be recovered by extraction only from that half of the animal in which it had been injected. There is therefore "storage" at the site of injection. The limits of storage after implantation of pellets were suggested by Forbes,[12] who stated that 90 per cent was absorbed in the rat in fifty-one days.

Toxicity

The dosage is properly limited to that which gives the desired effect, but also by possible gastrointestinal disturbances, and occasionally by less common sequelas, such as those noted in the literature by Morrell.[13] These include headache, drowsiness, vertigo and dermatoses. Von Haam et al.[10] mention excessively increased libido. We have not been informed of anything approaching this by our patients; nor indeed, except rarely, have we found it to increase sex drive at all. Physicians were made unduly apprehensive of the toxicity of stilbestrol by some early investigators, who, accustomed to the mild estrogenic effects of some of the hormones, used what proved to be unnecessarily large doses. The long delay in approval by the Food and Drug Administration, while it very properly gathered large numbers of reassuring reports,[6, 13–16] contributed to the uncertainty among clinicians.

Our experience regarding toxic reactions is much the same as those cited. We find that such symptoms as nausea, vomiting, headache and malaise were encountered in 31 of 199 patients (15.6 per cent) treated for various complaints, chief among which were menopausal symptoms and menstrual disorders.* Toxicity depends on individual sensitivity as well as on dosage. The menopausal patients appear to be less susceptible than those treated for menstrual disorders. Toxic symptoms were reported in only 12.4 per cent of the former, as compared with 23.1 per cent of the latter.† Pregnant or lactating women are said to tolerate tremendous amounts.[8, 15, 17, 18] Zondek and Bromberg[19] mention a patient who could take one hundred and seventy times as much stilbestrol when pregnant as when not pregnant; they concluded that there was a detoxication of the material during pregnancy. Toxic reactions are not confined to stilbestrol, but may be elicited also by overdosage with any of the hormonal estrogens.[6] There seems to be no evidence of serious liver, kidney or bone-marrow damage, such as was feared following the earliest reports,[13–16, 20] when unsuspectedly excessive doses were used. Transient nausea is the commonest of the infrequent objections to the drug. We have satisfactorily avoided this by instructions to take the tablet at bedtime, because the disagreeable symptoms usually follow only a few hours after ingestion. Abarbanel and Klein[8] find that bile salts or acid preparations help in preventing these effects. Others[21] believe that the enteric-coated pill is less troublesome.

Finch[22] regards these untoward symptoms as an allergic response and reports desensitization to the drug by frequent medication, beginning with a low dosage and gradually increasing this until the therapeutic level is reached. An allergic reaction to stilbestrol had previously been noted by Baird.[23]

Hamblen[24] expresses perhaps the greatest danger in the use of nonhormonal estrogens: "Being orally active, cheap and highly potent, uncritical use of them by physicians and self medication with them by patients would add, doubtlessly, to an already widespread contraphysiologic and empiric estrogenic therapy."

Carcinogenic Effect

Much loose thinking has prevailed regarding the carcinogenic properties of the estrogens, among the most potent of which one must place stilbestrol. Since its effect on the uterus and breast is similar to that produced by various natural estrogens, it seems more than likely that its potentiality to develop cancer is the same. But for cancer to develop, the unknown cancer factor is generally considered essential. On the other hand, once the cancer factor is activated by the intrinsic growth potential or by artificial growth stimulus, such as Gardner et al.[25] gave in the form of estrogen to male mice of a cancer strain, the tumor may then progress, even if the artificial stimulus is discontinued. Removal of an endogenous agent sometimes effectively halts the growth. This has been seen after castration in some cases of metastatic cancer of the breast, and also of cancer of the prostate. The great majority of experimental efforts to initiate cancer in various species, even including the monkey, have failed. Zondek[11] has mentioned several investigative attempts, some apparently successful, and also reported his failure to induce cancer by giving tremendous doses of estrogen over many months to members of his strain of rats, among 5000 of which no spontaneous cancer had ever been found. He recorded Engle and Smith's failure to produce

*This study was carried on at the Free Hospital for Women, Brookline, Massachusetts, with material generously supplied by the Winthrop Chemical Company, Incorporated, New York City.

†The previously quoted figure, 15.6 per cent, refers to the incidence of toxic symptoms in a group of 199 cases, which also included other conditions, such as pruritus and sterility.

cancer in 4 macaques and also Zuckerman's inability to cause cancer in monkeys by large doses of estrone over long periods. Zuckerman treated monkeys for a year with a total of 365,000 I.U. of estrone. Because 2 women, both of whom had received 50,000 I.U. of estradiol benzoate daily for twenty-four days, or a total of 1,200,000 I.U., showed no carcinoma, Zondek concluded that even extremely large doses of hormone did not cause carcinomatous changes of the uterus. He believes that large doses may even protect against the initiation of cancer because of the consequent inhibition of the growth effect of the anterior pituitary gland, as well as by the resultant hormonal castration. In 195 patients who received appreciable amounts, we have seen no cancer develop during the exhibition of stilbestrol or soon after its consumption. Because of the nature of estrogen metabolism, long-delayed effects of its use are not to be apprehended. This does not mean that cancer need not be kept constantly in mind, for it can develop in any female at any age. However, we do not subscribe to the notion that any estrogen per se is carcinogenic.

Clinical Use

Primary Amenorrhea

Stilbestrol, like the natural estrogens, is unpredictable as a precipitant of bleeding in cases of primary amenorrhea but, if given for two or more weeks, usually produces flow, either during the treatment or as a withdrawal phenomenon. The dose need not exceed 2 mg. daily; Palmer[20] used seven daily doses of 1 mg. each, followed by seven daily doses of 5 mg. Doses larger than 2 mg. may elicit unpleasant nausea or tenderness of the breasts. In susceptible persons, however, toxic symptoms may follow the exhibition of only 0.1 mg. daily. If a clinical *tour de force* is sought because of the temporary psychologic "lift" that a period of flow gives the defective girl, it may be more surely obtained by the simultaneous ingestion of 2 mg. of stilbestrol and the intramuscular injection of 5 to 10 mg. of progesterone on five successive days.

Secondary Amenorrhea

In the girl or woman whose germinal epithelium is active, but who has merely stopped maturing follicles, stilbestrol is much more useful. One milligram daily by mouth for fourteen to twenty-one days is almost certain to be followed, within four to ten days, by a flow that satisfies the patient. In a very few cases, a second spontaneous flow follows some weeks after treatment. If a succeeding series of treatments is started at the time of the bleeding, the flow is usually repeated. This may be kept up for months, but the likelihood of spontaneous recovery of the ability to ovulate is not thereby enhanced; we have observed, however, that it is not always destroyed.

Aperiodomenorrhea (Immeasurable or Anovulatory Menstruation, Uterine Dysfunction)

When flowing is irregular in incidence or prolonged, and sometimes when it is profuse, it may be brought to a semblance of normality again by repeated cyclic series of stilbestrol treatments, consisting of 0.5 to 1.5 mg. daily for arbitrarily selected periods of fourteen to twenty-one days. Succeeding periods of treatment are started with or soon after each period of flowing.

Menopausal Symptoms Incident to Normal or Artificial Climacteric

The greatest value of stilbestrol is in the treatment of these conditions. Initial dosage is properly about 1 mg. daily. When comparative comfort has been re-established, the dose should be diminished to the individually critical level, which may seem ridiculously low; occasionally, it can be increased without causing toxic symptoms. Natural remissions in the first ten years of the normal menopause are not infrequent, and at such times, medication should be reduced or omitted. Intermittent administration of any estrogen is necessary to relieve the anterior pituitary gland of undue inhibitory influences. There is as yet no reason to suppose that the carefully graded use of any estrogen during the menopause merely postpones the evil day when the unpleasant subjective symptoms must be suffered before stabilization is accomplished. Rather, it seems that final comfort without medication can be gradually attained by the use of smaller and smaller doses of an estrogen; and stilbestrol is a good one.

Of great significance during and after the menopause is the fact that stilbestrol stimulates proliferation of the endometrium and establishes therein the potentiality to bleed, and that this potentiality may be activated during treatment and will surely express itself after cessation of the drug, if it has been used for several weeks. This phenomenon is important mainly because it may not disturb the unwary clinician as much as it does the apprehensive patient. The first bleeding from cancer of any part of the uterus must not be overlooked. Careful examination and continued close observation for long periods after omission of the drug are essential.

Suppression of Lactation

We have not used stilbestrol to inhibit lactation. Davis and Boynton[15] report that if treatment with 5 mg. for eight to ten days is started promptly on

delivery, no engorgement occurs. If, on the other hand, the drug is withheld until after lactation is established, they state that the engorgement of weaning, although it does take place, is usually slight. Abarbanel and Klein[8] found that stilbestrol in doses up to 500 mg. did not affect established lactation in the nursing woman. Mendel, Goldman and Caire[27] noted primary suppression of lactation in 98 per cent of 55 patients. Many other workers[8, 17, 18] present similar results, and all mention the extreme infrequency of any unpleasant symptoms, even though such large doses are given as would certainly be toxic to the nonpuerperal patient.[19] Abarbanel and Klein[8] gave doses ranging from 250 mg. a day to 1500 mg. a week to pregnant and puerperal patients, and recorded no toxic effects except occasional dizziness.

Dysmenorrhea

For dysmenorrhea, a very common gynecologic disorder, estrogens, stilbestrol among them, have a very limited field, because to relieve the pain they must prevent ovulation. Thus, to upset what is frequently a delicately balanced mechanism in any girl is usually too high a price to pay. Sturgis[28] is right when he says that if 1 mg. of stilbestrol is given daily for twenty days, beginning a full week before the expected time of ovulation, the succeeding period of flow will be painless. He finds, furthermore, that the omission of treatment in the succeeding cycle permits the re-establishment of ovulation. Therefore, the procedure may occasionally be justified in the rare patient who apprehends that pain will materially interfere with some important performance — artistic, athletic, social or academic. Frequent or prolonged use of this method might seriously upset the endocrine balance that is necessary for normal cyclic ovulation.

Senile Vaginitis

In postmenopausal vaginitis, suppositories containing 0.5 to 1 mg. of the drug used daily for several weeks will rejuvenate the vaginal epithelium and re-establish its high resistance to the commoner bacterial and fungoid irritants. Davis and Boynton[15] recommend the use of a 0.5 mg. suppository each evening for six to eight weeks; we have noted alleviation of the condition in less time. Abarbanel and Klein[8] effectively used a stilbestrol ointment for this purpose, and also suggested stilbestrol in suppositories for gonorrheal vulvovaginitis in children, a condition with which we have had no experience.

Pruritus Vulvae

As an adjuvant in the relief of vulvar itch, which is often very distressful, stilbestrol by mouth in doses of 0.5 or 1 mg. daily, or by inunction, as reported by Abarbanel and Klein,[8] by Wharton[29] and by Lubowe,[30] may be very helpful, but other measures should also be taken. The urine must be sugar free. The application of 2 per cent boric acid helps to eradicate the almost constant bacterial dermatitis, and painting with gentian violet usually removes the commonly found fungus.

As already mentioned in connection with the menopause, stilbestrol may also evoke bleeding in the aged if given for several consecutive weeks. When it does so, the treatment must be stopped, and any recurrence should be regarded as indicating cancer until thorough search by curet and biopsy reveals no such growth.

* * *

Stilbestrol, a potent synthetic estrogen, is marketed in various forms, since it is effective orally, intramuscularly or percutaneously, and bids fair to supplant the natural estrogens in therapeusis. The standard daily dose is considered to be 0.1 to 1 mg., although some patients profitably take as much as 5 mg. Overdosage or undue susceptibility is associated with nausea and occasionally with various other symptoms of mild toxicity, but there is no evidence that the drug is dangerous. It has been reported that desensitization is possible by the use of gradually increasing doses. During pregnancy and lactation, tolerance is tremendously raised.

Like other estrogens, stilbestrol seems not to be carcinogenic per se, but it does cause proliferation in the epithelium of the reproductive system.

Its greatest usefulness is in the treatment of menopausal symptoms and in the production of flow in the amenorrheic patient. It may occasionally be used for the relief of dysmenorrhea, provided that treatment is started at least one week before ovulation time, so that follicular rupture is prevented.

References

1. Dodds, E. C., Goldberg, L., Lawson, W., and Robinson, R. Oestrogenic activity of certain synthetic compounds. *Nature* **141**:247, 1938.
2. Council on Pharmacy and Chemistry. Designations "stilbestrol" and "diethylstilbestrol" for the synthetic estrogen 4:4'-dihydroxystilbene and its diethyl derivative. *J. A. M. A.* **117**:1625, 1941.
3. Scaley, J. L., and Sondern, C. W. Comparative estrogenic potency of diethylstilbestrol, estrone, estradiol and estriol. II. Uterine and vaginal changes in infantile rats. *Endocrinology* **29**:356-362, 1941.
4. Lipschütz, A., and Vargas, L., Jr. Comparative action of stilbestrol and other estrogens on endometrium of the castrated guinea pig. *Endocrinology* **29**:622-627, 1941.
5. Lee, H. M., Robbins, E. B., and Chen, K. K. The potency of stilbestrol in the immature female rat. *Endocrinology* **30**:469-473, 1942.
6. Greene, R. R. Reactions to estrogens. *Am. J. Obst. & Gynec.* **42**:858-861, 1941.
7. Stoddard, F. J., and Metzger, I. A clinical comparison of three commercial estrogenic preparations. *J. Clin. Endocrinol.* **2**:209-212, 1942.
8. Abarbanel, A. R., and Klein, M. D. Clinical experiences with stilbestrol. *New York State J. Med.* **41**:383-389, 1941.
9. Greenhill, J. P. Recent advances in gynecologic endocrinology. *Illinois Med. J.* **79**:61-66, 1941.
10. von Haam, E., Hammel, M. A., Rardin, T. E., and Schoene, R. H. Clinical studies on stilbestrol. *J. A. M. A.* **115**:2266-2271, 1940.
11. Zondek, B. *Clinical and Experimental Investigations on the Genital Functions and Their Hormonal Regulation.* 264 pp. Baltimore: Williams & Wilkins Co., 1941.

12. Forbes, T. R. Absorption of pellets of crystalline testosterone, testosterone propionate, methyl testosterone, progesterone, desoxycorticosterone and stilbestrol implanted in the rat. *Endocrinology* 29:70-76, 1941.
13. Morrell, J. A. Summary of some clinical reports on stilbestrol. *J. Clin. Endocrinol.* 1:419-423, 1941.
14. MacBryde, C. M., Castrodale, D., Loeffel, E., and Freedman, H. The synthetic estrogen diethylstilbestrol: clinical and experimental studies (II). *J. A. M. A.* 117:1240-1242, 1941.
15. Davis, M. E., and Boynton, M. W. Indications, clinical use and toxicity of 4-4' dihydroxy-diethyl stilbene. *J. Clin. Endocrinol.* 1:339-345, 1941.
16. Mazer, C., Israel, S. L., and Ravetz, E. The synthetic estrogen stilbestrol: an experimental and clinical evaluation. *J. A. M. A.* 116:675-681, 1941.
17. Diddle, A. W., and Keettel, W. C., Jr. Clinical effect of stilbestrol on postpartum activity of the mammary glands. *J. Clin. Endocrinol.* 1:494, 1941.
18. Clahr, J. Suitable dosages of stilbestrol for suppression of lactation. *J. Clin. Endocrinol.* 1:759-762, 1941.
19. Zondek, B., and Bromberg, Y. M. Autodetoxication of stilbestrol during pregnancy. *Lancet* 1:381, 1942.
20. Grauer, R. C., and Nugent, E. Studies in diethylstilbestrol. II. Differences in response of radium induced and physiological menopause. III. Effect on liver. *Surg., Gynec. & Obst.* 74:686-691, 1942.
21. Gray, L. A., and Gordinier, J. D. The effect of diethylstilbestrol and diethylstilbestrol dipropionate on postmenopausal vaginitis and symptoms. *Am. J. Obst. & Gynec.* 41:326-328, 1941.
22. Finch, J. W. The nausea and vomiting following administration of diethylstilbestrol. *J. A. M. A.* 119:400-402, 1942.
23. Baird, K. A. Allergic reaction to stilboestrol. *Canad. M. A. J.* 44:401, 1941.
24. Hamblen, E. C. Uses and limitations of estrogens in gynecic practice. *J. A. M. A.* 117:2205-2207, 1941.
25. Gardner, W. U., Smith, G. M., Allen, E., and Strong, L. C. Cancer of the mammary glands induced in male mice receiving estrogenic hormone. *Arch. Path.* 21:265-272, 1936.
26. Palmer, A. Clinical experiments with diethylstilbestrol. I. Estrogen withdrawal bleeding in primary and postmenopausal amenorrheic women. *Am. J. Obst. & Gynec.* 41:861-867, 1941.
27. Mendel, F. B., Goldman, A. M., and Caire, A., Jr. Inhibition of lactation with stilbestrol. *Am. J. Obst. & Gynec.* 42:528-530, 1941.
28. Sturgis, S. H. The use of stilbestrol in the relief of essential dysmenorrhea. *New Eng. J. Med.* 226:371-376, 1942.
29. Wharton, L. R. Treatment of pruritus of the perineum, labia and vagina by newer methods, including injection of alcohol and the use of stilbestrol and an astringent cresol derivative. *Southern M. J.* 34:694-698, 1941.
30. Lubowe, I. I. The use of stilboesterol cream in the treatment of pruritus vulvae and allied disorders. *Urol. & Cutan. Rev.* 45:314, 1941.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28411

PRESENTATION OF CASE

A fifty-two-year-old married woman was admitted to the hospital because of pain in the chest.

Ten months prior to admission, the patient noticed transient pain in the chest beneath the left breast. This was not severe but was worse on inspiration. Soon after this, she developed exertional dyspnea and often wheezed. She was treated by her physician for a "heart condition," and digitalis was administered. Following this, all symptoms were relieved. Six weeks before entry, the patient developed a little pain under the upper portion of the left shoulder blade and occasional pains in the left shoulder. At about this time, she noticed that the left upper chest seemed "full" and that the veins over the chest were prominent. She believed that she had lost a little weight.

The family and past histories were irrelevant. A brother had died of cancer of the rectum.

Physical examination revealed a nervous, well-developed and well-nourished woman in no distress. The pupils were equal in size. Examination of the heart and right lung was negative. The left upper chest seemed fuller than the right. The veins over this side appeared prominent. The amplitude of inspiratory and particularly expiratory movements was reduced. The anterior and posterior aspects of the upper left chest were flat to percussion. Tactile fremitus and breath sounds were diminished above the fifth rib anteriorly and the eighth rib posteriorly. There was a fullness of the left supraclavicular fossa, which seemed to extend beneath the clavicle. The left hand was warm and dry; the right was cool and wet. Abdominal examination was negative.

The blood pressure in the left arm was 152 systolic, 82 diastolic, and in the right arm, 138 systolic, 80 diastolic. The temperature was 98.1°F., the pulse 78, and the respirations 20.

Examination of the blood revealed a hemoglobin of 14.5 gm. and a white-cell count of 8100 with 81 per cent polymorphonuclears. The urine was acid in reaction and had a specific gravity of 1.022; the sediment contained 22 white cells and 7 epithelial cells per high-power field. An electrocardiogram demonstrated a normal sinus rhythm of 65. There was a tendency toward right-axis deviation, but the record was within the normal range. T_1 was flat to very slightly diphasic; T_4 was flat to very slightly inverted. The blood Hinton test was negative.

An x-ray film of the chest revealed a dense homogeneous mass that filled the left pleural cavity from the apex to the eleventh rib (Fig. 1). The lower margin of the mass was lobulated. The mediastinum and heart were displaced markedly to the right. The lung between the lower limits of the mass and the left diaphragm appeared to be bright, probably owing to emphysema. The right lung field was clear, and there was an azygos lobe. There was no definite evidence of erosion of the ribs or enlargement of the intervertebral foramens. The patient was operated on fourteen days after admission.

DIFFERENTIAL DIAGNOSIS

DR. HELEN PITTMAN: May we see the x-ray films?

DR. GEORGE W. HOLMES: Whatever this process is, it has advanced beyond the stage where the radiologist can make a complete diagnosis. The process is homogeneous throughout on the routine film. On a film taken with a Bucky diaphragm, this finding was confirmed. Consolidation with cavity formation might look like this, on the first film, but not like that, on the second; so we can be fairly certain that the lesion had a fairly homogeneous density throughout. The heart and mediastinal contents are displaced away from the lesion. That is evidence of some space-filling lesion, such as fluid or tumor, rather than collapse. The fact that the lower part of the lung field is clear is against a large accumulation of fluid in the pleural space. The fact that it bulges might be interpreted as due to fluid or tumor. The absence of erosion of the ribs or bodies of the vertebras is against a tumor's arising from the sympathetic chains or the spinal nerve roots, — the neurofibroma group, — but it is not against a primary lung tumor. The fact that the other lung is clear is against a tuberculous lesion. I think the evidence we have here rather points toward neoplasm; whether it was primary or secondary, we have no definite evidence.

DR. PITTMAN: We are obviously faced here with a tumor of the mediastinum, and how much farther than that I can go I am not at all sure. I can do a certain amount of reasoning and a larger amount of guesswork, and end up with "tumor of the mediastinum, type to be determined later on."

We have a fifty-two-year-old woman, who was not sick, who may have lost weight, but who was still well nourished, and who had no fever or leukocytosis. Ten months before entry, she began to have pain. Then she was well for eight months but, by the time of entry, was having more symptoms. The only symptoms that she had had are sympathetic system, demonstrated by the fact that her hands showed different temperatures and different degrees of moisture, and bronchial pressure, indicated by lack of aeration except at the base, where there appeared to be some emphysema.

I am not going to inflict on you a complete differential diagnosis of tumors of this region of the

FIGURE 1. *Anteroposterior Film of the Chest.*

those that I attribute to pressure. True, the pain that she had at the beginning in her left breast and the pain later in the left shoulder may have been pleural and not due to pressure. A few things here I think are a little queer. If not queer, I just do not understand them. She had a large mass in the left chest bulging above the clavicle. Vascular compression was evidenced by dilated veins, and yet the blood pressure on that side was higher than on the other side. I have no idea how or why that occurred. She also had pressure on the body. I think there are three things to be considered.

The first one I thought of, as I read over the record and before I had seen the x-ray films, was aneurysm, and we still must consider aneurysm of the descending aorta; such lesions are relatively symptomless and likely to be silent until they have attained considerable size and are then picked up because of pressure symptoms. This holds true in this woman. On the other hand, with an aneurysm of this size, I think we should expect to find

some evidence of erosion of the vertebral bodies or ribs. The blood Hinton test was negative, and she had equal pupils. I do not believe we have enough evidence here to make a diagnosis of aneurysm.

Then we come to new growths in this region, and a solid malignant tumor is a possibility. I believe sarcoma is far and away the commonest. This patient had a very large lesion without any evidence, so far as we are told, of metastatic disease. It seems hard to me to consider sarcoma as large as this with no evidence of the same disease elsewhere in the body, and in a woman who is not sick. Lymphoma can never be excluded. One always has to mention it. Very much the same argument holds as in sarcoma. The only temperature report is a normal one. Again, the woman is not sick, and no mention is made of enlarged lymph nodes. It does not say that she had none, but I am assuming that that information is not intentionally left out, and that none were found.

I have never seen an intrathoracic thyroid gland at all comparable in size with this, and I am just not taking any stock in it. That brings us down to benign tumors of the mediastinum, which are statistically rare. I think, however, that a benign tumor fits in with the situation we have here reasonably well, a very large growth with no symptoms until it caused pressure, and in a woman who was not ill.

A dermoid is always a possibility. The Bucky film certainly shows a uniform density, but unless one can pick up a tooth or bone or something else of different density or unless there is ulceration into the bronchus, one cannot be sure. The patient coughed up no characteristic material. I do not see that we have any right to think we are dealing with a dermoid, particularly since this woman was older than most individuals are when dermoids are found. A lipoma, which could occur in this position, must be seriously considered even though it is not seen very often. I believe fibromas also can arise in this position. They are not seen often, but again they are picked up only because of their pressure symptoms in an essentially healthy person. I have considered this mass as having arisen from the structures of the mediastinum, but I suppose there is no way of being sure it did not arise from either bronchus or lung. We have very little, however, pointing to its origin in a bronchus or the lung, so I am going to stick to the mediastinum as the source. I am therefore left just exactly where I was in the beginning, with a large lump occupying the chest, and I do not know what it is, but I think it is extremely unlikely to be a malignant tumor. I think we have no data on which to make a diagnosis of aneurysm and I therefore make a diagnosis of benign tumor and suggest that it might have been a lipoma.

DR. TRACY B. MALLORY: Would you expect it to have that degree of density, Dr. Holmes?

DR. HOLMES: No.

DR. MALLORY: A fibroma would fit better with what you see?

DR. HOLMES: Yes.

DR. EDWARD D. CHURCHILL: We made a diagnosis of benign mediastinal tumor. The rest of the problem was a technical and mechanical one. The first consideration, in which we very likely decided incorrectly, was the route of approach. There were two choices. The incision may be placed anterolaterally or posterolaterally. Because of the evidence that the tumor extended above the clavicle, and in anticipation of difficulty in freezing it from the subclavian artery or vein, we chose an anterolateral incision. The operative notes read as follows:

> A portion of the seventh rib anteriorly was resected, and the pleural cavity opened. An enormous tumor was found, apparently completely filling the left chest. It was obvious that a wider opening of the chest was necessary. The sixth rib was divided and retracted. This did not give satisfactory exposure, and it was necessary to divide the manubrium, which was done transversely in the region of the sixth costal cartilage. This enabled a hand to be passed up and around the tumor. The lung was found displaced and compressed downward and backward, and apparently was completely atelectatic. The tumor had extended high into the left apex and was adherent to the posterior mediastinum and posterior surface of the left apex of the chest. It was freed up and gradually released. There was profuse bleeding from veins on the inner aspect of the chest wall in the apex. An attempt was made to stop hemorrhage by ligatures and suture. Several sutures were taken in the posteromedial aspect of the collapsed left upper lobe, and this in turn was sutured into the apex. During the procedure, the patient went into shock, apparently owing to blood loss. Two transfusions were given during the operation.

In order to deliver this enormous solid tumor even after making the maximum exposure by resection of ribs, it was necessary to divide the sternum transversely. I had hoped of course that the tumor was cystic and that its size could be reduced by aspiration, but that was impossible because it was a solid tumor. A tumor of this size in the chest does not have a pedicle, like a tumor of the ovary, which can be simply dealt with because, when it is delivered, the pedicle can be secured. This tumor had a broad base on the lateral aspect of the vertebral column and the heads of the ribs.

The patient died of primary hemorrhage because the blood was coming much faster than we could put it in with transfusion. This represents

an error in surgical judgment, since we could not control a situation that we had created. Whether, if I had utilized a posterolateral incision, it would have brought me nearer the lateral aspect of the vertebras with better opportunity to control the large veins before they were cut or torn, I do not know. If I were doing it again with this experience in mind, I should certainly use a posterolateral approach.

Clinical Diagnosis

Mediastinal tumor (? neurofibroma).

Dr. Pittman's Diagnosis

Benign tumor of mediastinum (? lipoma).

Anatomical Diagnosis

Fibroma of mediastinum.

Pathological Discussion

Dr. Mallory: From the histologic point of view, this was a fibromatous tumor, evidently very slowly growing and of long duration. It showed small areas of cystic degeneration, though not enough so that an appreciable amount of fluid could have been withdrawn and the mass significantly reduced in size. It was essentially a solid tumor. There were also large foci of xanthomatous degeneration with many cholesterol crystals. Whether it arose from the perineurial sheaths, I cannot say with certainty. It has not the obvious earmarks of perineurial fibroma, but on the other hand, there are areas that do suggest such a diagnosis. Since the majority of tumors in the mediastinum are of neural origin, that would be my guess; but I cannot prove it.

Dr. Churchill: There was a definite constriction ring in the tumor representing the first rib and the clavicle. It was hourglass in form where it had extended up above the first rib and had become tightly wedged in place.

Dr. Mallory: There is one discrepancy between your operative findings and the x-ray report. That is the question of air beneath the tumor. Your impression, I believe, was that the lung was entirely collapsed.

Dr. Churchill: Undoubtedly the lower portion of the lung contained air.

Dr. Ralph Adams: Would you expect erosion of the ribs posteriorly in such tumors?

Dr. Holmes: Yes.

Dr. Churchill: That was another reason we went in anteriorly.

Dr. Lerman: Can you tell where the blood supply is coming from?

Dr. Churchill: Only in retrospect.

Dr. Jacob Lerman: In any type of tumor?

Dr. Churchill: Very often it is predictable, as, for example, with an intrathoracic goiter.

CASE 28412

Presentation of Case

A thirty-six-year-old married woman was admitted because of pain in the chest.

Approximately three months prior to admission, the patient began to have rather severe aching and, at times, shooting pains in the left side of the chest above the breast. Often, the pain seemed to radiate down the left arm and occasionally to the spine posteriorly. The episodes varied in length from a few minutes to an hour and occasionally awakened the patient during the night. The pain was not related to exertion. During this illness, the patient suffered with infrequent dizzy headaches and was slightly dyspneic. She was able to climb only a single flight of stairs. Occasionally, the dyspnea was relieved by taking soda and belching. During this time, she raised a small amount of pale-yellow sputum without coughing. Two months before entry, x-ray treatment was administered to the chest, following which her symptoms of shortness of breath were relieved and the amount of sputum reduced.

The family history was noncontributory. When twenty-six, the patient had pneumonia, which was complicated with right-sided empyema necessitating rib resection and open drainage; Type 4 pneumococci were recovered from the pus. She made a satisfactory recovery and had no symptom related to the chest until the present trouble.

Physical examination revealed an obese woman in no acute distress. Examination of the heart and lungs was negative except for a few rales audible at the base of the right lung posteriorly. There was a slight amount of tenderness over the entire lower abdomen — the patient stated that she was menstruating.

The blood pressure was 110 systolic, 70 diastolic. The temperature was 99°F., the pulse 77, and the respirations 20.

Examination of the blood revealed a hemoglobin of 14 gm., and a white-cell count of 9500 with 73 per cent polymorphonuclears, 15 per cent lymphocytes, 6 per cent monocytes, 5 per cent eosinophils and 1 per cent basophils. A catheterized specimen of urine was acid in reaction, had a specific gravity of 1.015 and gave a ++ test for albumin; the sediment showed 20 white cells, occasional epithelial cells and many bacteria per high-power field. The blood Hinton reaction was negative. The nonprotein nitrogen was 22 mg. per 100 cc.

An x-ray film of the chest demonstrated an 8-by-5-cm. homogeneous mass attached to the right posterior margin of the trachea from the level of the clavicle downward; the mass displaced the trachea and esophagus to the left (Fig. 1). The displacement of the esophagus appeared to be due to pressure without evidence of attachment. The mass moved with swallowing. In the lateral view, there appeared to be some narrowing of the trachea in the region of the mass. The fifth and sixth right ribs showed old resection, with partial regeneration of the bone in the posterior axillary line. There was evidence of old pleurisy on the right. The lung fields were clear. The heart appeared normal.

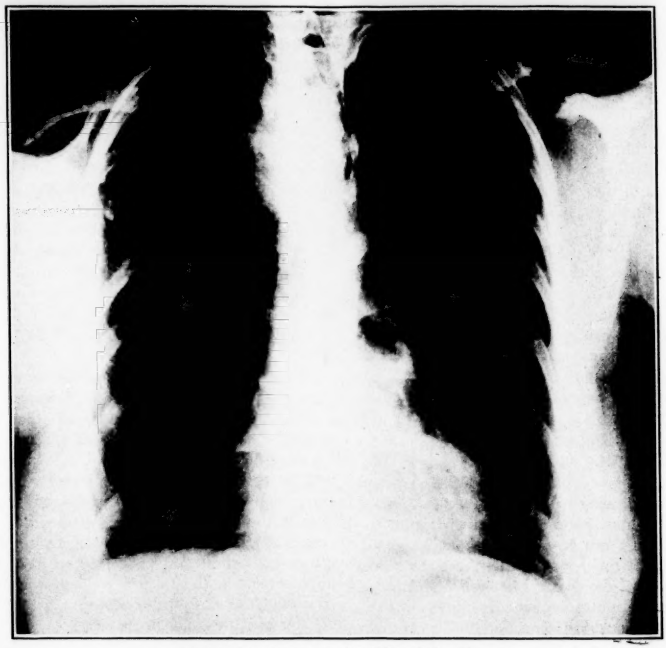

FIGURE 1. *Anteroposterior Film of the Chest.*

Eight days after admission to the hospital, an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. RALPH ADAMS*: May we see the x-ray films?

DR. GEORGE W. HOLMES: The patient had had part of a rib removed. This is the mass that we are interested in. It shows most clearly in this second film, taken with a Bucky diaphragm to bring out the outline of the tumor. Here you see the displaced esophagus and trachea. The arch of the aorta is here. The lateral view does not help us.

DR. ADAMS: Does the displacement of the trachea extend high enough to reach the level of the cartilages of the neck?

DR. HOLMES: I think it does.

DR. ADAMS: But does it go up to the level of the thyroid cartilage?

DR. HOLMES: It is questionable whether it does that.

DR. ADAMS: Is the esophagus displaced anteriorly or posteriorly? Or can you tell?

DR. HOLMES: Not from these films, and I do not believe anything is said about it in the notes.

DR. ADAMS: It states in the records, "The displacement of the esophagus appeared to be due to pressure without evidence of attachment." Could you rely on the member of your department who made that statement?

DR. HOLMES: I suspect it was Dr. Richard Schatzki. He has been doing a lot of work on that subject, and I should put considerable weight on what he says. How he finds it out, I do not know.

*Surgeon, Lahey Clinic.

DR. ADAMS: The problem in differential diagnosis in this case is to define the type of superior mediastinal tumor that is displacing the esophagus and trachea, causing dyspnea by tracheal compression and producing pain by usurping space at the thoracic inlet, where space is limited. Mediastinal tumors are unsurpassed as stimulators of diagnostic guessing bees, and rarely can the correct guess be supported by critical evidence logically establishing the one diagnosis and definitely excluding all others.

From the history one obtains nonspecific information of both positive and negative value. Radiation of pain to the midclavicular line is observed in many pulmonary and mediastinal lesions, perhaps because anterior cutaneous branches of the intercostal nerves are distributed to that region. The intermittent character of the pain is indicative of an expanding lesion, but expansion can occur in either a cystic or a solid growth. The increasing dyspnea can best be explained by pressure and distortion of the trachea, which was compressed anteroposteriorly and displaced laterally to the left. Other possible explanations, such as heart disease or lung disease, are ruled out by lack of collateral symptoms and physical or x-ray signs of disease in the heart or lungs.

The pale-yellow sputum produced without coughing is puzzling. Most sputum is coughed up. In fact, only sputum lying in the upper trachea and larynx can be raised without coughing. The absence of cough means that there was no irritative lesion of the tracheal or bronchial mucosa, and yet the accumulation of small amounts of pale-yellow sputum, probably mucoid in type, rather than purulent, suggests that there was some mucosal irritation. Extrinsic pressure on the upper trachea could produce this paradoxical situation. Decrease in sputum and dyspnea after x-ray treatment might mean that a malignant tumor, such as lymphoblastoma, had proved sensitive to irradiation, had shrunken in size and had been followed by clinical improvement. It could also mean that the x-ray therapy had merely suppressed secretory function of the tracheal mucosa and that the improvement in dyspnea was coincidental. There is nothing in the clinical summary suggestive of carcinoma of the esophagus or bronchus, that is, no difficulty in swallowing, no food regurgitation, no cough and no hemoptysis: some one of these symptoms would surely be associated with a carcinoma of the bronchus or esophagus casting an x-ray shadow of this size. Also, x-ray films show none of the features to be expected in an invasive lesion of the esophagus or trachea. Cancer will therefore be dropped from further consideration.

It is contrary to usual experience to find a lymphoma of this size localized to one portion of the mediastinum without palpable nodes in the neck, axillas or groins, and with a blood smear so nearly normal. A lymphoma that responded to x-ray treatment with regression in symptoms should have almost disappeared within two months if the treatment was adequate. A tumor of the lymphoblastoma series is therefore believed unlikely.

The history of pneumonia and empyema apparently has no connection with this episode and presumably is included to explain the rib defects and the old pleurisy at the right lung base.

The temperature of 99°F., the white-cell count of 9500 and the polymorphonuclear percentage of 73 indicate slight infection. One possibility is cystitis, because of the report of urine containing albumin and white cells. Another is that the lesion in the chest contained infected material or was surrounded by an inflammatory reaction.

Mediastinal dermoids commonly arise in the anterior portion of the chest, and if they attain sufficient size, most often displace the midline structures posteriorly. This lesion displaced the trachea anteriorly. A dermoid cyst probably is not the answer, although it is ruled out on this point alone. Mediastinal neurofibromas tend to arise in the posterior regions, in proximity to the sympathetic nerves. Consequently they appear in the costovertebral gutter, and before resulting in displacement of the trachea and esophagus, cause erosion of the ribs over which they lie. There is no sign of rib erosion in this case.

Except for the statement that "the displacement of the esophagus appeared to be due to pressure without evidence of attachment," one would consider seriously the diagnosis of a benign tumor of the esophageal wall, such as a fibroma, neurofibroma or esophageal cyst. This statement undoubtedly was recorded by the X-ray Department after careful fluoroscopic study with barium, and must be accepted as final. Approximately a hundred benign tumors of the esophagus have been reported since 1717, and the diagnostic features have been fairly clearly defined. It is improbable that the x-ray staff of this hospital would have missed such a diagnosis.

Any mass of the superior mediastinum that moves with swallowing is probably a substernal goiter, on a purely statistical basis. Substernal goiters, unless they are examples of goiter in aberrant thyroid tissue, always have an attachment to the lower pole of the thyroid gland. In consequence, the tracheal deviation they cause begins

at the thyroid cartilage and extends downward to the lower margin of the substernal extension, being maximal at the line of greatest tumor diameter. There is only an exception of 1 per cent to this rule. In the case under discussion, the tracheal deviation did not begin at the thyroid cartilage, but lower at the level of the first thoracic vertebra. Again, substernal goiters descend anteriorly to the trachea, but there is an exception of 10 per cent to this rule. However, 95 per cent of all goiters lying behind the trachea within the chest cause palpable and recognizable irregularity within the neck. Since the neck of this patient was normal, the combined statistical likelihood that she had a substernal goiter becomes 0.05 per cent, leaving it a possibility but not a probability.

A so-called "bronchiogenic cyst," arising from the right posterior margin of the trachea, could produce the symptoms and findings here recorded, and this is my diagnosis.

DR. EDWARD D. CHURCHILL: I might have been comforted if I had known these accurate statistical chances of the patient's having a substernal goiter, because, not knowing what the mass was, my chief worry was whether it could best be approached through the neck or through the chest. I did not want to operate through the chest and find a substernal goiter. It also crossed my mind that this might have been a parathyroid tumor lying in the posterior mediastinum. High-lying posterior mediastinal tumors can in general be delivered very readily through a cervical incision. I decided on the transthoracic approach and was correct, because this mass was intimately adherent to the lateral aspect of the trachea throughout its whole extent and could not have been exposed from above. It had to be separated from the trachea by sharp dissection, which in some areas exposed the mucous membrane of the trachea.

CLINICAL DIAGNOSIS

Mediastinal tumor (? neurofibroma).

DR. ADAMS'S DIAGNOSIS

Bronchiogenic cyst.

ANATOMICAL DIAGNOSIS

Bronchiogenic cyst, arising from trachea.

PATHOLOGICAL DISCUSSION

DR. MALLORY: I have the specimen here. It was cystic, filled with very thick, partially inspissated, mucoid material. In its walls, numerous plaques of cartilage could be easily felt. On microscopic examination, it is evident that it is lined with ciliated epithelium and that it contains numerous mucous glands, cartilaginous plaques and occasional bundles of smooth muscle, in other words, all the various structures that go to make up the trachea or bronchi. Therefore, it can be classed as a bronchiogenic or tracheal cyst, as Dr. Adams predicted.

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CESAREAN SECTION IN MASSACHUSETTS

The five-year study of cesarean sections in Massachusetts, sponsored by the Massachusetts Department of Public Health and the Section of Obstetrics and Gynecology of the Massachusetts Medical Society and supervised by Dr. Robert L. DeNormandie, has been completed, and a summary of the results appears in this issue of the *Journal*. The compilation and analysis of even a limited amount of data on such a large series of cases is an arduous task, and Dr. DeNormandie and his associates deserve a large measure of credit for carrying it through to completion.

The report necessarily falls far short of being a thorough statistical study, but it furnishes considerable prima-facie evidence of abuse of this operation throughout the State. How extensive this abuse is, in view of the limited information available, must be a matter of speculation. The two most frequent indications given for primary section were contracted pelvis or disproportion and dystocia. There is no way of knowing precisely what was meant by "dystocia." The term simply means difficult childbirth. Presumably what was meant in most cases was so-called "cervical dystocia" or labor which, though prolonged, was ineffectual in dilating the cervix. These two indications combined account for 3234 cesarean sections, practically 1 per cent of the total births. It hardly seems plausible that in 1 out of every 100 cases of childbirth in Massachusetts, a section is necessary because the pelvis is too small, the baby too big or the labor ineffective. The third most frequent indication for primary section was placenta previa, of which there were 1021 cases. If all these patients had placenta previa, the incidence of that complication was about 1 in 327 births, and if there were any patients with placenta previa who were not delivered by cesarean section, the incidence was, of course, correspondingly greater. Such a frequency of placenta previa is scarcely credible. If the repeat sections are excluded, it appears that in 330,464 births there were 7763 primary sections — an incidence of about 1 in 43. The mere perusal of the indications given for some of these sections is sufficient to arouse suspicion. In 150 cases, they are, to judge by the samples given, utterly ridiculous, and in at least 170 others, they are manifestly indefensible.

The mortality in this series of cesarean sections is extraordinarily low — 2.5 per cent. A low mortality, however, is not an unmixed blessing, because it is a contributory cause of the reckless use of the operation. With a mortality risk of this order of magnitude, it is not at all improbable that even a busy physician will practice many years without having a death follow cesa-

rean section, and undoubtedly many a physician is much more influenced by his personal experience than by a statistical fact. When the mortality of cesarean section is between 1 and 2 per cent, the operation is not more hazardous than are some of the more difficult and complicated pelvic deliveries, and if the operator in a difficult or complicated pelvic delivery is not especially trained in obstetrics, section is probably less hazardous. When, however, a healthy woman who could be delivered simply and easily through the pelvis is subjected to cesarean section, her chance of dying is multiplied about ten times. This is a truth that certainly is not widely known among the laity and seems not to be comprehended by many physicians.

The extensive use of cesarean section is not, of course, so great a factor in total maternal mortality as it is often said to be. A large proportion of all maternal deaths follow cesarean section, but this is due, in part at least, to the fact that the operation is often employed to deliver patients with such serious complications as heart disease, nephritis, severe pre-eclampsia and placenta previa. These patients run a much greater risk than normal patients, regardless of how they are delivered. During the five years covered by this study, the total maternal mortality dropped from 4.1 to 2.9 per 1000 live births. The incidence of cesarean section, however, remained practically constant, and the slight drop in cesarean mortality is quite insufficient to account for the drop in total mortality. What is needed is not simply fewer cesarean sections, but better obstetrics, and the really disturbing feature of this report is not the number of sections done, nor the frequency of foolish indications, but the indirect evidence that there is still a great deal of bad obstetrics being done in Massachusetts.

MASSACHUSETTS MEDICAL SERVICE

As of Tuesday, October 6, 1058 doctors had become participating physicians in the Blue Shield.

OCCUPATIONAL SKIN DISEASE

Wartime industrial activities have provided the Nation not only with a host of victims killed and permanently disabled by injury but also with much disability because of cutaneous damage. Not everyone is aware of the fact that 65 per cent of disease due to occupational factors concerns the skin, or that the consideration of occupational liability may arise in a fifth of all cases with cutaneous involvement, especially if the hands and arms alone are affected. Furthermore, the amount of disability produced by the effects of industrial processes on the skin has increased in the last few months, owing, in all probability, to employees in new and unfamiliar occupations, perhaps inadequately trained in the cutaneous hazards involved and the proper care of the skin, to the speeding up of industrial processes, to the introduction of new ingredients and, perhaps, to other factors. Particularly disturbing are the reports of increased irritation of the skin by cutting oils used in the manufacture of planes, tanks and guns. Numerous other examples of cutaneous hazards in war industries are cited by Downing elsewhere in this issue of the *Journal*.

For the medical profession, the early detection, the proper treatment and the prevention of occupational dermatoses constitute a real challenge. A review[1] of the recent report of the Committee on Industrial Dermatoses of the Section on Dermatology and Syphilology of the American Medical Association defines the criteria for the recognition of these diseases, and these points are further discussed in a recent issue of the *Journal*.[2] Proper treatment requires recognition of the causal factor and the development of proper protective measures or the elimination of that factor; and it implies the avoidance of presumably therapeutic

substances that may exaggerate an already inflamed skin. 20 to 25 per cent of all cases of dermatitis are due to some form of therapy. In prevention, however, lies the real answer. The working out of prophylactic measures against a particular cutaneous hazard is often difficult, but much progress has been made, as is indicated in Schwartz and Tulipan's[3] recent textbook on the subject and in recent bulletins issued by the United States Public Health Service.

Occupational dermatoses are for the most part preventable. In these days, it is the duty of every physician to contribute to the war effort by recognizing these cases, by returning such patients to work at the earliest possible moment and by preventing further disability to them and other employees.

References

1. Lane, C. G., Dennie, C. C., Downing, J. D., Foerster, H., Oliver, F. A., and Sulzberger, M. Industrial dermatoses. *J. A. M. A.* 118:613-615, 1942.
2. Lane, C. G. Recognition of occupational dermatoses. *New Eng. J. Med.* 227:39-43, 1942.
3. Schwartz, L., and Tulipan, L. A. *Text-Book of Occupational Diseases of the Skin.* 799 pp. Philadelphia: Lea & Febiger, 1939.

MEDICAL EPONYM

Romberg's Sign

Moritz Heinrich Romberg (1795-1873), professor and director of the Royal Polyclinic Institute of the Friedrich-Wilhelm University, Berlin, described this sign in his *Lehrbuch der Nerven-Krankheiten des Menschen (Textbook of Human Nervous Diseases)*, (Berlin, 1851: Vol. 2, p. 185). A portion of the translation follows:

> If the patient is asked to close his eyes while standing upright, he immediately begins to sway and reel. . . . I called attention to this pathognomonic sign ten years ago (according to my observation, it does not occur either in other paralyses or in uncomplicated blindness) and have since never failed to find it in any of my numerous patients with this disease.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

A Plea for Breast Feeding

The war emergency has precipitated many changes in activities for the public good. Perhaps no more important contribution, particularly in view of the threatened shortage of physicians, can be made for this and the coming generation than by renewing the efforts to urge mothers to nurse their babies.

In their textbook, *Diseases of Infancy and Childhood*, Holt and McIntosh state:

> Maternal nursing is the natural and ideal method of infant feeding. Every mother should nurse her infant unless there are weighty reasons to the contrary. The large majority can do so. The physician should do all in his power to encourage maternal nursing and to insure its success.

This statement of facts cannot be more succinctly expressed. The depleted ranks of the medical profession, the increase in the birth rate and the modified and often unfavorable conditions for infant care emphasize the importance of the above statement and the duty of the physician in this emergency.

Lactation can be promoted by the practice of the accepted principles of prenatal care, with emphasis on a well-balanced diet. And, of great importance in establishing lactation are skillful obstetric care and the proper education of nurses in the technic of breast feeding. Psychology plays no little role, especially in the early stages of lactation, and the nurse, by her skill and encouragement, is often the determining factor.

The technic of nursing consists in regularity of feedings, preferably every four hours on both breasts, five or six times daily. Complete emptying of the breast stimulates the secretion of milk, and a normal infant secures its quota of milk in twelve minutes of active nursing. Although the duration of each nursing cannot be empirically stated, it certainly is undesirable to urge babies to nurse beyond a twenty-minute period.

A common error is to omit nursing because the baby has not gained according to certain standards. A stationary weight or a slow gain for a week is often followed by an adequate supply of mother's milk. Furthermore, vomiting and loose stools are too often attributed to "poor milk." In general, a mother's milk is suitable for her infant, and these symptoms are only rarely of importance.

Since over 80 per cent of all babies in Massachusetts are born in hospitals, it is the responsibility of the nursing staffs and the visiting physicians to institute all the procedures that are necessary to ensure maternal nursing.

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College of Physicians and Surgeons, Boston, 1929. Sponsor: G. Lynde Gately, 624 Bennington Street, East Boston.

CALICCHIO, ELIA J. A., 240 Hanover Street, Boston.
College of Physicians and Surgeons, Boston, 1928. Sponsor: Whitman K. Coffin, 438 Marlboro Street, Boston.

CORRADO, JOHN C., 104 St. Andrew Road, East Boston.
Middlesex University School of Medicine, 1933. Sponsor: G. Lynde Gately, 624 Bennington Street, East Boston.

CURRY, JOHN J., Evans Memorial Hospital, Boston.
Tufts College Medical School, 1938.

DEVINE, JOSEPH W., 773 Broadway, South Boston.
College of Physicians and Surgeons, Boston, 1931. Sponsor: P. F. Butler, 35 Bay State Road, Boston.

DINATALE, PETER, 498 East Broadway, South Boston.
Tufts College Medical School, 1939.

FORZIATI, ANTHONY A., 35 Washington Avenue, Winthrop.
Middlesex University School of Medicine, 1932. Sponsor: George H. Schwartz, 19 Princeton Street, East Boston.

HITE, JACOB W., 51 Park Drive, Boston.
Middlesex University School of Medicine, 1930. Sponsor: Louis E. Wolfson, 113 Bay State Road, Boston.

KARP, ISADORE A., 153 Shurtleff Street, Chelsea.
Middlesex University School of Medicine, 1936. Sponsor: Frederick Ornsteen, 131 Washington Avenue, Chelsea.

MEYERS, MARVIN T., 8th Medical Battalion, Fort Jackson, South Carolina.
Kansas City University of Physicians and Surgeons, 1931. Sponsor: Robert Slater, 587 Beacon Street, Boston.

MORRISON, PHILIP J., 66 Beacon Street, Boston.
Harvard Medical School, 1939.

SPRINZ, HELMUTH, 226 Hemenway Street, Boston.
University of Berlin, 1936. Sponsor: Joseph H. Pratt, 30 Bennet Street, Boston.

TAUBER, MAX, 185 Chambers Street, Boston.
Middlesex University School of Medicine, 1935. Sponsor: Nathan N. Levins, 30 Chambers Street, Boston.

Hollis L. Albright, *Secretary*
412 Beacon Street, Boston

WORCESTER DISTRICT

BRAHM, LEO, 188 Highland Street, Worcester.
University of Berlin, 1924. Sponsor: Frank H. Washburn, Holden Clinic, Holden.

COX, ROBERT D., Belmont Hospital, Worcester.
Tufts College Medical School, 1938.

DAY, HARRY L., City Hospital, Worcester.
Yale University School of Medicine, 1934.

FADGEN, JOHN F., 29 Pierce Place, Clinton.
Loyola University School of Medicine, 1939.

GLASS, SARAH E., Worcester State Hospital, Worcester.
Rush Medical College, University of Chicago, 1938.

GRZYB, HENRY A., 151 Hamilton Street, Southbridge.
College of Physicians and Surgeons, Boston, 1937. Sponsor: Percy A. Brooke, 27 Elm Street, Worcester.

LEVINE, IRVING M., Grafton State Hospital, North Grafton.
Tufts College Medical School, 1939.

YASUNA, ELTON R., 50 South Lenox Street, Worcester.
New York University College of Medicine, 1938.

ZUCKER, JOSEPH M., Worcester State Hospital, Worcester.
Johns Hopkins University School of Medicine, 1936.

Julius J. Tegelberg, *Secretary*
390 Main Street, Worcester

WORCESTER NORTH DISTRICT

ARGOFF, JOSEPH, 813 River Street, Fitchburg.
Missouri College of Medicine and Science, 1927. Sponsor: Thomas R. Donovan, 42 Fox Street, Fitchburg.

WYANT, JOHN A., 101 Prichard Street, Fitchburg.
College of Physicians and Surgeons, Boston, 1936. Sponsor: Alverne P. Lowell, 52 Hartwell Street, Fitchburg.

Edward A. Adams, *Secretary*
44 Oliver Street, Fitchburg

DEATH

MIKOLAITIS — CASIMIR JOHN MIKOLAITIS, M.D., of Lawrence, died July 7. He was in his fifty-ninth year.

Dr. Mikolaitis received his degree from the Chicago College of Medicine and Surgery in 1914. He was a former member of the Massachusetts Medical Society.

WAR ACTIVITIES

OFFICE OF CIVILIAN DEFENSE

SUPPLEMENTAL STAFFS FOR EMERGENCY BASE HOSPITALS

Selected hospitals and medical schools in the coastal states have been invited by the Surgeon General of the United States Public Health Service to organize affiliated staff units that will be ready to serve when needed to supplement the medical staffs of emergency base hospitals, now being designated by the Medical Division of the Office of Civilian Defense. These units resemble the affiliated hospital units of the Army except that they are smaller. They are being organized to assure suitable status and remuneration for physicians who may be called on in the event of an enemy attack in their locality to care for casualties and other patients who have been evacuated to the interior of their region.

The designation of emergency base hospitals and the formation of affiliated units are part of a joint hospital program of the Medical Division of the Office of Civilian Defense and the United States Public Health Service. The program is authorized under an agreement concluded March 2, 1942, between the Federal Security Administrator and the director of the Office of Civilian Defense.

Physicians in the affiliated units will be commissioned in the inactive Reserve Corps of the Public Health Service. Unless an urgent need for their services should arise, they will remain on an inactive status for the duration of the war. They will be called to active service only if hospitals in their regions must be evacuated and the civilian population must be moved because of military necessity. Activation of the units will take place by order of the Surgeon General at the request of the chief medical officer of the Office of Civilian Defense on advice of

the regional medical officer and the state chief of Emergency Medical Service in charge of the affected areas.

The commission will be in grades ranging from passed assistant surgeon to senior surgeon, and when units are activated, these officers will have the rank, pay and allowances equivalent to those of officers in the armed forces.

Institutions invited to form units are asked to nominate an outstanding physician or surgeon as unit director, who, if he meets the physical and other requirements, will be commissioned senior surgeon in the Public Health Service Reserve. The unit director will then nominate the remainder of the staff, and appointments will be made after clearance through the state chief of Emergency Medical Service. Nominations are to be limited to male physicians over forty-five years of age, to those under that age who have physical disabilities that disqualify them for military service but that do not interfere with their professional activities, and to women physicians.

To avoid serious depletion of the professional staffs in the medical schools and hospitals of the target areas, the Surgeon General has recommended that medical schools draw their affiliated units in part from associated hospitals and that nonteaching hospitals invite physicians from other qualified hospital staffs to collaborate.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1942

Diseases	August 1942	August 1941	Five-Year Average*
Anterior poliomyelitis	2	45	44
Chicken pox	121	125	106
Diphtheria	12	6	11
Dog bite	1077	998	1099
Dysentery, bacillary	4	29	20
German measles	83	31	25
Gonorrhea	479	283	401
Measles	296	281	287
Meningitis, meningococcal	19	3	5
Meningitis, other forms	2	4	—†
Mumps	271	273	150
Paratyphoid infections	13	9	19
Pneumonia, lobar	121	74	100
Scarlet fever	224	195	118
Syphilis	354	341	391
Tuberculosis, pulmonary	303	260	297
Tuberculosis, other forms	24	24	30
Typhoid fever	10	8	10
Undulant fever	4	13	4
Whooping cough	673	575	520

*Based on figures for preceding five years.
†Pfeiffer bacillus meningitis only other form reportable previous to 1941.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Braintree, 1; Weymouth, 1; total, 2.

Diphtheria was reported from: Athol, 1; Cambridge, 1; Fall River, 3; Lowell, 1; New Bedford, 1; Somerset, 1; Somerville, 1; Springfield, 1; Woburn, 2; total, 12.

Dysentery, bacillary, was reported from: Boston, 1; Danvers, 1; Lowell, 1; Salem, 1; total, 4.

Encephalitis, infectious, was reported from: Weston, 1; Weymouth, 1; total, 2.

Lymphocytic choriomeningitis was reported from: Worcester, 1; total, 1.

Malaria was reported from: Boston, 1; Camp Edwards, 3; Fort Banks, 1; Quincy, 1; total, 6.

Meningitis, meningococcal, was reported from: Boston, 6; Brockton, 1; Cambridge, 2; Dedham, 1; Fort Devens, 1; Fitchburg, 2; Plymouth, 1; Revere, 2; South Hadley, 1; Springfield, 1; Worcester, 1; total, 19.

Meningitis, other forms, was reported from: Natick, 1; Revere, 1; total, 2.

Paratyphoid infections were reported from: Amherst, 4; Arlington, 1; Leominster, 1; Lowell, 1; Methuen, 1; Southwick, 1; Tyringham, 1; Woburn, 2; Worcester, 1; total, 13.

Septic sore throat was reported from: Boston, 1; Lynn, 1; Milton, 1; Waltham, 1; Worcester, 1; total, 5.

Tetanus was reported from: Holyoke, 1; total, 1.

Trachoma was reported from: West Boylston, 1; Worcester, 1; total, 2.

Typhoid fever was reported from: Arlington, 1; Beverly, 1; Cambridge, 2; Lynn, 1; Quincy, 1; Rockland, 1; Worcester, 3; total, 10.

Undulant fever was reported from: Cambridge, 1; Clarksburg, 1; Fitchburg, 1; Townsend, 1; total, 4.

Communicable diseases reported at figures above their five-year averages were chicken pox, diphtheria, German measles, gonorrhea, measles, pneumonia, scarlet fever, pulmonary tuberculosis, whooping cough and mumps. The last disease had been previously reported at record-high figures for eleven of the preceding twelve months.

Meningococcal meningitis was reported at a record-high figure.

Anterior poliomyelitis reached a record-low figure during this month.

Diseases reported at lower figures than their five-year averages include dog bite, bacillary dysentery, paratyphoid infections, syphilis and tuberculosis, other forms.

Typhoid and undulant fevers were both reported at figures equal to their five-year averages.

Two cases of animal rabies were reported, one from each of the foci in the southeastern and northeastern sections of the State.

CORRESPONDENCE

APPROVED LABORATORIES FOR PREMARITAL BLOOD TESTS

To the Editor: The list of laboratories approved for performing premarital blood tests has recently been revised. There are eight such laboratories on the present list, in addition to the Wassermann Laboratory of the department. These are:

Boston
- Boston Dispensary
- Boston Health Department
- Leary Laboratory
- Massachusetts General Hospital
- Peter Bent Brigham Hospital

Brockton
- Health Department

New Bedford
- St. Luke's Hospital

Springfield
- Mercy Hospital

Several other laboratories have made application for approval. It is likely that others will be added to the list about January 1.

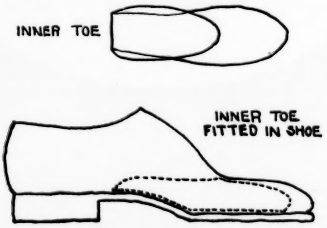
Paul J. Jakmauh, M.D.
Commissioner of Public Health
State House
Boston

INNER TOE FOR UNDERSIZED FOOT

To the Editor: Many persons, because of poliomyelitis or some accident in youth, have one shorter and more or less underdeveloped foot. The problem of suitable footwear for such mismated feet is very burdensome.

A shoe of a pair bought to fit only the larger foot will give little support or comfort to the smaller foot, which shifts about loosely within it. In a short time, the toe of this shoe will begin to curl up and form a deep crease behind the toecap, which is unsightly. Shoes of different sizes may be bought or be made to order at considerable expense but even these, although more comfortable, are somewhat too noticeably unmated.

To some of these persons, an inner toe, a drawing of which is appended, and which can be transferred from shoe to shoe like an arch-support, will entirely solve this problem. It has been made for me by George H. Lapham and Company, of Boston, at very small cost, and consists of thin leather, entirely similar to that of the ordinary men's house slipper. It is, in fact, a house slipper made to order for the smaller foot but cut off at a point short of the heel, where the edge, under the instep, cannot be felt by the wearer. It fits rather snugly into the toe of a stock-model shoe and is held in place without pads or stuffing.



It seems to wear indefinitely. It entirely prevents any curling up or creasing of the toe of the shoe and gives all the support and comfort of a shoe made to order.

ALLAN R. CUNNINGHAM, M.D.

76 Church Street
Winchester, Massachusetts.

COMMITTEE ON INDUSTRIAL HEALTH

To the Editor: I am sorry to ask that space in the *Journal* be used for the enclosed ill-natured correspondence.

The stand of the Committee on Industrial Health is stated in the first paragraph of my letter to Dr. Muller. If Dr. Muller will inform the committee how it can tell a prospective employer the race, color and religion of an applicant, without knowing this ourselves, or, if he will tell us how we can exempt physicians from the requirements frequently exacted of all other employees in industry in Massachusetts, we shall be grateful to him. We do not believe that the second paragraph of Dr. Muller's letter of September 26 would be helpful in trying to evade the questions asked in the personnel offices.

DWIGHT O'HARA, M.D., *Chairman*
Committee on Industrial Health

* * *

To the Editor: The Committee on Industrial Health of the Massachusetts Medical Society sent out questionnaires concerning availability of physicians for industrial appointments. In the upper corner are five questions, three of which do not belong there: "race," "color" and "religion."

To answer the first it would be necessary to know if the committee is using the groups according to Dr. Hrdlicka or Mr. Hitler. Even then, I cannot see how much influence "race" would have on the usefulness of a physician. Color or religion certainly makes no more difference.

As an old member of the Society and a humble naturalized citizen, may I use the pages of the *Journal* for recommending the Constitution of the United States as required reading to the gentlemen on the Fenway.

JOSEPH MULLER, M.D.

36 Pleasant Street
Worcester, Massachusetts

This letter was referred to Dr. O'Hara, Chairman of the Committee on Industrial Health. ED.

Dear Dr. Muller:

You probably do not appreciate that the Committee on Industrial Health is attempting to secure doctors who will be satisfactory to employers, as well as employment that will be satisfactory to doctors. As the relation between doctor and patient is no less personal in industrial than in other types of practice, employers may be very much interested in the data called for. For this reason, the data are pertinent and purposeful.

I think your letter is both unpleasant and uncalled for. For this reason we are refraining from publishing it, together with comments which we might make upon it.

If you will look at your Selective Service registration card, you will see that the Government of the United States is also interested in knowing your race. It is respectfully suggested that if you wish to register with the Committee on Industrial Health, you may use the same grouping that you did for the United States of America.

DWIGHT O'HARA, M.D.

Dear Dr. O'Hara:

I agree that my letter was unpleasant; it is certainly not uncalled for. The reason you do not want it published, is that your reply does not answer my question, but evades it.

Your statement that race is registered on the draft registration card is true, but the question about color is the colloquial way used in the United States for this division of the human race. As your questionnaire contains the question of race and color, your answer is not entirely to the point. If you were interested in the question of race, as it is understood by scientific American anthropologists, putting in a question about race and color was redundant, and hardly worthy of a professor of one of our great universities.

You failed to answer my inquiry why the registrant's religion is of any importance. I think that your action in preventing my criticism to appear in print, is more worthy of Mr. Petrillo and his breed, than of an officer of the Massachusetts Medical Society.

In case you would have posted your questionnaires immediately, you would also know that I sent in my questionnaire ahead of my letter, for in these times personal criticisms and animosities cannot interfere with any of the duties which legally constituted authorities impose on the citizen.

JOSEPH MULLER, M.D.

REPORTS OF MEETINGS

BOSTON CITY HOSPITAL

The fifth of a series of lectures on war medicine sponsored by the Boston City Hospital House Officers' Association was delivered by Dr. Louis Wolfson, recently of the American Hospital in Britain, who discussed "Injuries of the Face and Neck."

Although 80 per cent of the injuries seen in base hospitals just outside large cities were in civilians, they are also a good indication of the type of wounds received in other areas. There were four large groups of accidents: bombings, directly or from debris; severe burns, especially those of the hands and face of aviators in "bailing out" of flaming machines; those associated with blackouts, which accounted for about one quarter of all plastic-surgery cases; and those sustained by the expeditionary forces returning from Dunkirk in boats. In certain cases, these hospitals are the site of first treatment, as in aviators, who are congregated immediately at one hospital. Usually, however, the plastic-surgery cases have had first-aid and even definitive treatment elsewhere, and are seen at late stages.

Time is of the utmost importance in the healing of the hand and face burns of aviators. It has been demonstrated that the use of saline baths three or four times daily, followed by dusting with sulfonamide powder and a minimal amount of handling, will reduce the time of convalescence 50 per cent over that required when escharotics were employed. In the subsequent plastic handling of the face scars, ectropion is first treated. In all cases, pedicle grafts are given precedence over Thiersch grafts.

A recent development in the management of fractured mandibles that shows promise is the use of Kirschner wires incorporated in plaster. This method allows chewing in twenty-four to seventy-two hours, and a more rapid healing of the bone ensues.

First-aid treatment of face wounds should be undertaken with the knowledge that this region is remarkably resistant to infection and has fine recuperative powers because of its unusually rich blood supply. Skin flaps, therefore, should not be sacrificed during the process of wound excision unless they are beyond hope. Approximation of the skin edges should always be attempted but should never be carried out under tension. If tension exists, it is better to approximate as well as possible and then use adhesive strapping. Undermining of the skin edges invites infection and affords poor cosmetic results. Sutures should be of the finest available material, with small bites, and very close. Ligation of bleeders is seldom necessary and actually contraindicated when pressure suffices. For this purpose an Ace bandage is unsurpassed. Cleansing should be carried out with the knowledge that foreign bodies such as glass and clothing are not revealed by roentgenograms.

All fractures around the face should be treated immediately, for it is then easier to reduce them, and the cosmetic results are much better. This includes the nose and jaws. In the latter, wiring is carried out immediately if possible. Fractures involving the sinuses have a higher incidence of infection and, consequently, poorer results. These are all drained, preferably internally if that is at all feasible. Plastic surgery is postponed for at least six months in these cases, and the bone of choice for grafting is the iliac crest. All deep wounds around the face are drained at least with catgut. Perforating lacerations involving the buccal mucosa have sutures taken in both surfaces. Burns of the face are treated with sulfanilamide powder after the usual cleansing. These drugs are also being used with apparent impunity on skin grafts.

HARVARD MEDICAL SOCIETY

A regular meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on April 28, with Dr. James P. O'Hare presiding. The usual presentation of cases was omitted. The speaker of the evening was Dr. Timothy Leary, medical examiner of Suffolk County, who discussed "The Genesis of Atherosclerosis."

Since geriatrics is becoming an increasingly important subject, and since the treatment of atherosclerosis is not particularly effective, the value of prophylaxis is apparent. And for the accomplishment of that end, more definite knowledge of causation is essential.

The crucial form of arteriosclerosis is atherosclerosis, which is distinguished from other forms by the presence of fatty material in the form of cholesterol esters in the lesions. Aschoff's theory of causation is that wear and tear from blood pressure injures the deep layers of the arterial intima. Since this layer depends for nutrition on the diffusion of plasma from the blood circulating in the artery, injury to the deep layers is followed by degeneration and the precipitation of free cholesterol esters from the plasma. The free esters are picked up by phagocytic cells, which become lipoid (foam) cells. Klotz objected that in early lesions there was no free cholesterol esters and no degeneration; the foam cells appeared first in the subendothelial layer and not in the deep layers. He believed that the Aschoff theory was based on the study of advanced lesions in which free esters arose from the breaking down of foam cells. Dr. Leary then pointed out how his investigations support the observations of Klotz.

The Russian school produced atherosclerosis-like lesions in rabbits by feeding animal fats and later by feeding cholesterol. This work, however, was not accepted, and the disease was disparagingly called "the cholesterol disease of rabbits."

Cholesterol, although present in combined form in all animal cells, normally occurs in visible form, as seen with the polariscope, only in the adrenal cortex and the myelin sheaths and sometimes in testicular interstitial cells and corpora lutea. Otherwise, visible cholesterol is associated with pathologic conditions. It is literally excess cholesterol, and experimental evidence indicates that this excess is a chronic irritant, stimulating growth of connective tissue.

The early lesions of atherosclerosis are made up of foam cells. In atheroma, the reversible condition in youth, the excess cholesterol is removed from the arteries by fibroblastic cells, and the lesions disappear. But in progressive processes, such as atherosclerosis, the cholesterol accumulates and provokes fibrosis, and the lesions enlarge into typical atheromatous nodules.

In rabbits, there is a latent period of weeks following the beginning of feeding before lesions appear in the aorta. By sacrificing animals at critical intervals during this period, Dr. Leary has shown that the ingested cholesterol is brought to the liver and esterified; that the esters collect in excess in the liver and adrenal glands; that Küpffer cells in the liver and similar cells in the adrenal cortex engulf the excess esters as particulate matter; that these ameboid cells, now foam cells, escape from the organs through the blood and lymph streams, pass the so-called "lung filter," and invade the aortic intima. In this way, atherosclerosis is produced. The disease, whether human or experimental, cannot occur in the absence of excess cholesterol.

Cholesterol is synthesized in the human body. The excess is probably ingested in the form of egg yolk, milk and animal fats. Needs for this substance are greatest in times of rapid cell formation. Egg yolk is intended for the embryo, and milk for the nursling. Metabolism of cholesterol is most efficient in the young, but heredity is certainly a factor. The tissue content of cholesterol increases with age, whereas the need for it grows less. Women mobilize this substance during pregnancy, possibly for the use of the embryo, and show minor mobilization during menstruation. They are less susceptible to atherosclerosis than men.

Excess cholesterol, then, is the cause of atherosclerosis, but stresses determine the localization of the lesions. Sex and thyroid secretions influence cholesterol metabolism, and age and heredity are modifying factors. Diets with limited or absent cholesterol should prevent atherosclerosis, and this may be accomplished by the substitution of vegetable oils, whose sterols are not absorbed, for animal fats.

The lesions of coronary sclerosis are almost exclusively atherosclerotic. The frequency of coronary disease is related to the unusual stresses to which the vessels are subjected. These vessels arise directly from the aorta in a region where there is maximal stress when the aortic cusps close. Moreover, circulation is cut off from the muscular branches by compression as the ventricles contract. At the same time, the main vessels and their subepicardial branches are not compressed, but are filled with blood under systolic pressure, without being able to empty themselves. The main coronary circulation is during diastole. Because of these stresses, normal coronary arteries develop characteristic thickening of the intima to produce a "buffer layer."

Advanced coronary sclerosis in patients as young as twelve years indicates that atherosclerosis is not entirely a disease of old age. But the type of lesion varies with age. In youth, excess cholesterol provokes a growth of loosely textured fibroblastic tissue. As the lesions enlarge, diffusion of nutritive material from the circulating blood becomes inadequate, and necrosis of the deep layers results. When the lesions become even larger, vascularization by capillaries from the lumen, or less commonly from the vasa vasorum, supplies the nutriment. All advanced coronary lesions are vascularized. The lumen is narrowed by eccentric or concentric thickening of the intima. Spasm may result in rupture of capillaries, fibrin formation and fibrinoid necrosis. With extension of the necrosis to the endothelium, thrombosis follows, and is the usual cause of sudden death.

In middle age, more collagen is formed, and scarring is typical of this period. Narrowing of the lumen, with coronary insufficiency and chronic vascular myocarditis, is frequently found at post-mortem examination. Spasm may result in sudden death or in infarction without anatomic occlusion of the vessels, particularly in hypertensive heart disease. Thrombosis is less common than in youth, whereas calcification becomes commoner.

In old age, atherocheumas occur frequently because of lowered tissue reaction to excess cholesterol. Occlusion may be caused by rupture of such a lesion. Scarring persists from earlier periods, and calcification is practically the rule.

Rupture through the necrotic tissues of a cardiac infarct may cause death by hemopericardium and cardiac tamponade. Rupture of an atherosclerotic coronary artery may likewise lead to hemopericardium. Dissecting aneurysms of the coronary arteries are rare. Infarcts are repaired by granulation, sometimes with pericardial adhesions. Mural cardiac thrombi may lead to systemic embolism. The scar tissue repairing an infarct may undergo stretching, producing a cardiac aneurysm. Coronary thrombi undergo organization if the patient lives, sometimes with canalization. But, usually, the blood flow is re-established around the region of thrombosis by a collateral circulation. Anastomoses between coronary arteries increase with age. In conclusion, it was pointed out that coronary occlusion may be symptomless, or may cause mild or serious illness; it may result in sudden death with ventricular fibrillation or death so instant as to suggest immediate cerebral arrest with cardiac and respiratory standstill.

The discussion was opened by Dr. Alan Moritz, who questioned the existence of coronary spasm. But Dr. Leary replied that coronary infarcts without thrombosis can be demonstrated at autopsy and that death sometimes follows characteristic manifestations of coronary occlusion when minor post-mortem lesions are present, if any. Dr. Samuel Levine pointed out that in a study of 100 young patients with angina pectoris, 96 were males. He added that this indicates a strong endocrine factor; however, the localization of the atheromatous lesion seems to be a familial factor, possibly dependent on some congenital anatomic peculiarity. Dr. Leary suggested that the prevalence of the proximal portion of the left coronary artery as a site for a single thrombosis may be explained on the basis of the sharp angulation of this vessel and its main branch, which create a mechanical hazard in a person who is unable to handle cholesterol adequately.

In answer to questions from the floor, Dr. Leary stated that the blood cholesterol levels in experimental rabbits, as well as in patients with hypothyroidism, are elevated. At autopsy, the amount of atherosclerosis is roughly proportional to the amount of hypothyroidism, whereas hyperthyroid patients definitely have less or no atherosclerosis. The changes described in the liver and adrenal glands in the rabbit have not been observed in man. The relatively enormous accumulations of excess cholesterol in these organs in the experimental rabbit, whose normal diet includes no cholesterol, account for this difference. Lesions due to intermittent accretions of excess cholesterol over a period of years in man may be reproduced in weeks or months in the rabbit.

BOOK REVIEW

Neuroanatomy. By Fred A. Mettler, M.D., Ph.D. 4°, cloth, 476 pp., with 337 illustrations, including 30 in color. St. Louis: The C. V. Mosby Company, 1942. $7.50.

This is a new and valuable addition to the textbooks of neuroanatomy. The first part deals with the gross aspects of the neural systems, and the second, with microscopic anatomy. The text is clear, concise and simple in terminology. The illustrations, some in color, are a particular feature of the book. The drawings are excellent and the labeling adequate, and the figures are well correlated with the text. Adequate references to the literature are given, and the index is generous.

The author is to be congratulated on the quality of his product. In a field already covered by many other books, he has succeeded in writing a text that is superior to most. The format and press work are well fitted to the text. The book is worthy of the highest endorsement.

(Notices on page x)